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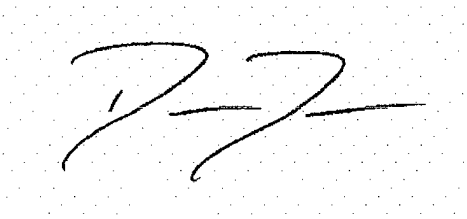

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Abstract

Title of Dissertation: Anxiety Sensitivity and Psychological Vulnerability

Darin R. Lerew, Ph.D., 1999

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To date, primary research in the area of panic disorder and similar anxiety pathology has been laboratory-based. A wealth of primary research in panic disorder in particular has been gleaned from laboratory research using biological challenge paradigms by which panic is experimentally provoked. The present work reviews the knowledge based gleaned from the biological challenge paradigm and the competing (and often agreeing) models of panic and anxiety, narrowing to a focus on Reiss' (1991) expectancy theory. Within expectancy theory, emphasis is placed upon the role of anxiety sensitivity. Expectancy theory proposes that anxiety sensitivity may serve as a premorbid risk factor for the development of anxiety pathology (Reiss, 1991). Next, this work presents a series of five reports investigating the role of anxiety sensitivity and psychological vulnerability in a number of areas. In addition, the concept of psychological vulnerability factors in general is explored, and other possible risk factors for anxiety pathology, depression, disability, and impairment are examined. The first three reports presented here stem from a large sample of data collected at the U.S. Air Force Academy (USAFA) during Basic Cadet Training (BCT) in the summer of 1995. The remaining two studies examine the theoretical position of anxiety sensitivity in terms

of pathology specificity (Schmidt, Lerew, & Joiner, 1998) and other more distal effects of elevated anxiety sensitivity such as decreased cardiovascular fitness (Schmidt, Lerew, Santiago, Trakowski, & Staab, under review).

The current studies are discussed in terms of the practical implications of findings regarding anxiety sensitivity, the relationship between vulnerability and impairment, and other potential vulnerability factors. In addition, the status of expectancy theory (Reiss, 1991) is discussed as a whole and comments are made concerning the ongoing debate regarding anxiety sensitivity and its relationship to trait anxiety. Data suggest that primary prevention of anxiety pathology may be possible with a focus on malleable vulnerability factors.

ANXIETY SENSITIVITY AND PSYCHOLOGICAL VULNERABILITY

By

Darin R. Lerew

Dissertation submitted to the Faculty of the
Department of Medical and Clinical Psychology
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Introduction

Anxiety is an innate, adaptive mechanism that readies human beings for action and protects them from anticipated threat. Unfortunately, this "alarm system" can become maladaptive when it is triggered for excessive lengths of time, triggered in situations known to be harmless, or when it is triggered for no apparent cause (Hoehn-Saric & McLeod, 1988). Such is the case with panic disorder and other anxiety pathology. The current paper reviews the status of panic and related anxiety pathology research in terms of laboratory and longitudinal studies, and presents a series of five manuscripts which report on one important component of anxiety termed "anxiety sensitivity." The meaning of these studies is discussed in light of panic and anxiety research as a whole.

The Phenomenology and Epidemiology of Panic Disorder

A panic attack is described as a discrete period of intense fear or discomfort accompanied by four or more somatic and/or cognitive symptoms (e.g., sweating, fear of dying) (American Psychiatric Association; APA, 1994). These symptoms develop abruptly and reach a peak within a 10-minute period. Panic attacks are seen across the spectrum of anxiety disorders, and are commonly seen in patients diagnosed with a specific phobia or social phobia. A formal diagnosis of panic disorder is warranted when an individual experiences recurrent, unexpected panic attacks followed by at least one month of persistent concern about having additional attacks, worry about the implications of additional panic attacks, and/or a significant change in behavior related to the attacks. The additional diagnosis of agoraphobia (i.e., panic disorder with agoraphobia) is considered when an individual exhibits significant avoidance of or distress associated with places or situations from which escape might be difficult or embarrassing, or in

which help may not be available in the event of a panic attack (APA, 1994). This disorder can significantly impair an individual's life in terms of social, occupational, and family functioning (Markowitz, Weissman, Ouellette, & Leib, 1989; Nagy, Krystal, Woods, & Chang, 1989).

Operationally, panic may be conceptualized as a tripartite system, consisting of physical (e.g. palpitations), subjective (i.e., fear), and behavioral (i.e., avoidance) symptoms. Although only four of thirteen symptoms are required for a diagnosis, patients generally report experiencing many more than this during panic attacks (Barlow & Craske, 1988). Diagnostically, it is important to note that some who experience full panic also report experiencing limited symptom attacks (less than four symptoms). Summarizing a series of studies using clinical samples, Barlow and Craske (1988) identified dyspnea, sweating, fear of going crazy & fear of losing control as the most commonly reported symptoms, and palpitations, dizziness, and fear of dying as the symptoms experienced most intensely.

Data from epidemiological studies such as the Epidemiological Catchment Area (ECA) study suggest that, over a lifetime, as many as 30-40% of all individuals will experience clinically significant anxiety (Shepherd, Cooper, Brown, & Kalton, 1966), approximately 28-34% will experience isolated panic attacks (Norton, Harrison, Hauch, & Rhodes, 1985), and between 1.5-6% will meet diagnostic criteria for panic disorder with and without agoraphobia (Eaton & Keyl, 1990; APA, 1994). At any given time, 3 percent of the adult population in the United States report recurrent panic attacks and 10 percent report occasional or isolated attacks (Weissman, 1988; Wittchen, 1986). The onset of panic disorder in treatment populations tends to occur in the late teens through

the late 20s (Barlow, 1988). Those afflicted with panic disorder suffer from negative personal, social, and economic consequences of a magnitude equal to or greater than that evidenced in major depression and serious medical conditions (Telch, Schmidt, Jaimez, Jacquin, & Harrington, 1995). Other sequelae include extremely high utilization of health services, increased risk for various medical conditions such as cardiovascular disease, and marked elevations in suicide attempts (Weissman, Klerman, Markowitz, & Ouellette, 1989; Markowitz, Weissman, Ouellette, Lish, & Klerman, 1989). Panic disorder is the most common problem reported by those who seek mental health treatment (Boyd, 1986; Klerman, Weissman, Ouellette, Johnson, & Greenwald, 1991), and the fifth most common complaint in primary care settings (Beitman, Basha, & Flaker, 1987; Klerman, Weissman, Ouellette, Johnson, & Greenwald, 1991). Untreated, the course of panic disorder is typically chronic (Keller & Baker, 1992).

Panic Etiology

Biological Theories

Due to the consistency with which the various challenge agents and procedures provoke panic, speculation has increased on the possibility of a neurobiological explanation for panic and several theories have been developed. Biologically oriented researchers have explained the effectiveness of the challenge procedures by asserting that they have a direct panic inducing effect and that individuals who panic during such procedures are neurochemically impaired (Clark, 1993). One recent and popular physiological theory of panic is that put forward by Klein (1993). Klein's false suffocation alarm theory posits that an innate suffocation alarm with a pathologically low trigger threshold is the core of panic disorder etiology. Klein (1993) postulates that the

faulty suffocation alarm is triggered both by rising levels of CO_2 in the brain and by situations that signal probable suffocation. Such an alarm could explain CO_2 inhalation-induced panic. In support of this theory, Klein (1993) argues that patients with panic disorder commonly report dyspnea during fear reactions. On the other hand, nonclinical controls typically report cardiovascular rather than respiratory symptoms during fear reactions (McMillian & Rachman, 1988). Also supporting the suffocation alarm theory, biological challenge agents that produce physiological correlates of asphyxiation (e.g., carbon dioxide) are reliably panicogenic in patients with panic disorder (Hollander, Liebowitz, Fyer, Gorman, & Klein, 1989: Levin et al., 1987; Woods, Charney, Goodman, & Heninger, 1988).

While carbon dioxide challenge studies support Klein's (1993) theory, they also raise questions regarding its validity. For example, according to the theory, carbon dioxide levels must rise to a level that mimics suffocation for the alarm to fire. Biological challenge literature suggests that this crucial level must be close to the level reached during carbon dioxide challenges, which equates to either one inhalation containing 875 times as much carbon dioxide as normal air (e.g., a single inhalation of 35% carbon dioxide) or 20 minutes continuous breathing of air containing 125 times as much carbon dioxide as normal air (i.e., 5% carbon dioxide). However, an individual (e.g., someone with panic disorder) is unlikely to encounter such high levels of carbon dioxide in everyday life (McNally, 1994). Furthermore, levels such as this should cross even the normal threshold of controls, however nonclinical controls rarely panic in response to carbon dioxide inhalation (Gorman et al., 1994). Another complication of Klein's theory is the suggestion that the level of internal carbon dioxide needed to trigger the alarm

exhibits both intra- and interindividual variation. In a test of the suffocation alarm theory, Schmidt, Telch, and Jaimez (1996) compared the responses of patients with panic disorder to a hyperventilation challenge and to a carbon dioxide challenge. Klein's theory predicts differential responding to these challenges, since one lowers arterial carbon dioxide levels (hyperventilation) and one raises carbon dioxide levels (carbon dioxide inhalation). Schmidt et al. (1996) reported findings inconsistent with this differential effects hypothesis, where no indicators of the suffocation monitor predicted differential emotional responding to the two challenges. Thus, Factors other than a hypersensitive suffocation alarm could account for differences between patients with panic disorder and other patients or nonclinical controls.

In part due to Redmond's (1977) work with monkeys and research with yohimbine, Charney et al. (1990) point to dysregulation in the noradrenergic system, specifically in the locus ceruleus, as a model for panic disorder. Caffeine acts primarily in the adenosine system but also indirectly stimulates the locus ceruleus, bolstering the argument for the role of both the noradrenergic and adenosine systems. The evidence for the noradrenergic model is mixed (McNally, 1994). Redmond's (1977) findings with monkeys have failed to be replicated in rats (Mason & Fibiger, 1979), drugs that effectively treat panic such as selective serotonin reuptake inhibitors do not directly affect noradrenergic receptors (den Boer & Westenberg, 1990), and the locus ceruleus serves many functions other than panic (van Dongen, 1981). Critics have pointed out that drugs that increase locus coeruleus firing (e.g., Buspirone, Carbamazepine) should be profoundly anxiogenic, yet actually have mild anxiolytic effects (Taylor, Eison, Riblet, & VanderMaelen 1985; Cohn & Wilcox, 1986; Uhde, Roy-Byrne, Vittone, Boulenger, &

Post, 1985). Furthermore, administration of Mianzeron, a drug which blocks the alpha-2 autoreceptor may relieve anxiety in some patients rather than producing panic (Klein, Rabkin, & Gorman, 1985). Clonidine's failure as an effective treatment for panic attacks also argues against the locus coeruleus modes, since it reduces locus coeruleus firing and should therefore be markedly anxiolytic (Hoehn-Saric, Merchant, Keyser, & Smith, 1981; Liebowitz, Fyer, McGrath, & Klein, 1981).

Peripheral beta-adrenergic receptor hypersensitivity has been considered as a possible mechanism in the etiology of panic disorder (Rainey et al., 1984). In early research, the similarity of symptoms of patients with beta-adrenergic hypersensitivity and patients with panic disorder has been noted by several authors (Frohlich, Tarazi, & Duston, 1969; Easton & Sherman, 1976), and infusion with isoproterenol, a beta-adrenergic agonist, produces panic more often in patients with panic disorder than in nonclinical controls (Rainey et al., 1984).

Nutt, Glue, and Wilson (1990) have hypothesized that patients with panic disorder may be characterized by dysfunction in the gamma-aminobutyric acid-benzodiazepine submolecular complex. Flumazenil, a benzodiazepine receptor antagonist assumed to be anxiolytic, was found to produce panic in patients with panic disorder (Woods, 1991). To explain this discrepancy, Nutt et al. (1990) postulated a receptor shift in panic disorder as the cause. They suggested that the "set point" in panic disorder patients' benzodiazepine receptors is shifted in the inverse agonist direction. With such a "shift" in receptor functioning, antagonists (e.g., Flumazenil) would act like inverse agonists and the effects of agonists (i.e., benzodiazepines) would be diminished. The high dosage of high potency

benzodiazepines required to treat panic disorder is consistent with such a hypothesis (Nutt et al, 1990).

Other hypothesized mechanisms of action in panic disorder include carbon dioxide chemosensitivity (Lousberg, Griez, & van den Hout, 1988), pH sensitivity (Papp et al., 1989), and serotonergic hypersensitivity (Targum & Marshall, 1989). In all of these models, a paucity of data precludes firm conclusions (McNally, 1994).

Psychological Theories

While biologically oriented researchers interpret data harvested from challenge paradigm research as indicating that challenges directly provoke panic via neurobiological pathways, an interpretation of psychologically oriented researchers is that challenges provoke panic by producing bodily sensations that panic patients interpret as signs of catastrophe (McNally, 1994). Despite the differences in the myriad panic provocation studies, two common factors emerge across all of them. First, a high level of baseline anxiety is a consistent predictor of panic (Ehlers, et al., 1986; Liebowitz, et al., 1984), and second, all of these challenges (with different physiological consequences) share the capacity to produce intense, uncontrollable bodily sensations capable of triggering fear (Barlow, 1988; McNally, 1994). As opposed to biological theories, psychological theories implicate these intense somatic sensations and their mediation by cognitive processes in the etiology of panic. Psychological models of anxiety focus on cognitive appraisal and parameters that affect the appraisal process. Below, three influential models of panic disorder are reviewed: Barlow's (1988) emotion-based model, Clark's (1986) cognitive model, and Reiss' (1991) expectancy model.

Barlow's Emotion-Based Model

Barlow's (1988) model of panic and panic disorder describes panic primarily from an emotion theory perspective but incorporates cognitive, learning, and biological aspects. Barlow (1988) views panic and anxiety as fundamentally different and provides specific definitions for each term (Antony & Barlow, 1996). Believing that fear is a distinct, basic emotion, Barlow (1988) describes fear as an alarm reaction consisting of an intense desire to escape from potential danger and both physical and cognitive mobilization for action. When the emotion of fear occurs in the absence of threat, the reaction is a "false alarm" or panic attack. Panic then, is identical to the emotion of fear (Antony & Barlow, 1996). On the other hand, anxiety is a more diffuse affect which varies across people and situations. For Barlow, anxiety is future-oriented apprehension over some possible threat whereas panic is an acute reaction to an immediate threat (Antony & Barlow, 1996).

According to Barlow (1988), the etiology of panic begins with a biological vulnerability toward being neurobiologically overreactive to stress. In addition to possessing a biological vulnerability, Barlow (1988) proposes that certain individuals possess an additional psychological vulnerability to developing panic disorder, consisting of a sense that events and emotions are uncontrollable and unpredictable. This sense results in an increase in arousal and an inward shift in attention, thus beginning a process of anxious apprehension regarding additional attacks. Individuals with anxious apprehension about future attacks have a propensity to associate interoceptive cues with the original false alarm through classical conditioning (i.e., "learned alarms"; Barlow, 1988). Learned alarms may then be triggered by specific bodily sensations, and with anxiety focused on future panic, "additional somatic and cognitive cues become available

to trigger the panic attacks, resulting in the development of panic disorder" (Antony & Barlow, 1996; p. 60). Conscious appraisal of sensations is not necessary for panic attacks, although Barlow's (1988) model acknowledges that some attacks may be preceded by the appraisal of danger regarding bodily sensations.

Clark's Cognitive Model

Clark's (1986) cognitive model of panic, like Barlow's, implicates bodily sensations as an important factor in panic disorder architecture. However, this model does not suggest a specific biological vulnerability (though it does allow for such a vulnerability). Clark's model of panic proposes that all panic is triggered by bodily sensations that are catastrophically misinterpreted as threatening. Catastrophic misinterpretation involves interpreting sensations as much more threatening than they really are. For example, an individual might perceive slight breathlessness as evidence of respiratory failure and consequent death (Clark, 1986). This perceived threat leads to more bodily sensations, greater perceived threat, and the cycle is repeated until the apprehension rises to panic. The misinterpreted bodily sensations may come from a variety of events, both emotional (e.g., anxiety related palpitations) and non-emotional (e.g., ingestion of caffeine). These panic-triggering sensations may also change across time depending on which bodily sensations are noticed and which fears the individual has been able to discount (Clark, 1986).

Clark (1986) notes that biological factors may play a role in panic. He states that they might increase an individual's vulnerability to panic in various ways. For instance, biological factors may contribute to the triggering of an attack if they cause the individual to experience more or more intense fluctuations in body state than others. For example, a

diabetic may be prone to panic due to fluctuations in body state associated with fluctuations in blood sugar. Also, biological factors may influence the extent to which a perceived threat produces an increase in bodily sensations. For example, a deficiency in the alpha-2-adrenergic autoreceptor would cause an individual to experience larger than normal surges in sympathetic nervous system activity in response to a perceived threat.

Reiss' Expectancy Model

Reiss and McNally (1985) and Reiss (1991) proposed that panic attacks, phobias, and other fear reactions arise from three fundamental fears: fear of negative evaluation, injury/illness sensitivity, and anxiety sensitivity. Fear of negative evaluation refers to apprehension and distress about receiving negative evaluations from others, expectations that others will provide negative evaluations, and avoidance of evaluative situations. Injury/illness sensitivity refers to fears of injury, illness, and death (Reiss, 1991; Taylor, 1993). Anxiety sensitivity is the fear of anxiety symptoms arising from beliefs regarding the consequences of experiencing anxiety (Reiss, 1991). In support of this theory, studies have shown that simple phobics and agoraphobics report that they are frightened by stimuli such as harmless animals or air travel because of fears of anxiety, illness/injury, or negative evaluation (Gursky & Reiss, 1987; McNally & Louro, 1992; McNally & Steketee, 1985). In line with Reiss' theory, these three fears have been shown to be factorially distinct and minimally intercorrelated (Taylor, 1993). Most relevant in research regarding panic disorder is the fundamental fear of anxiety sensitivity which is discussed in more detail below.

Other Psychological Theories of Anxiety

Although the focus of the current work is, generally speaking, that of cognitive theories of anxiety, it should be pointed out that several other psychological theories of anxiety have been proposed, and have been influential, over the years. Psychoanalytic theory has given rise to several theories beginning with that of Freud. Freud (1959) presented a number of different conceptions of anxiety, proposing first that anxiety results from the transformation of the affect of a repressed impulse. Thus, sexual frustration or the practice of *coitus interruptus* could produce anxiety. Later, Freud (1964) essentially reversed the causation in this chain of events, with repression being the consequence rather than the cause of anxiety. Rooted in the origins of sexual development, Freud's (1959, 1964) conceptualizations of anxiety still share some common ground with today's cognitive conceptualizations. For example, Freud explained that anxiety is an adaptive function, serving to mobilize "defenses" in an effort to prevent a dangerous situation from becoming a "traumatic" situation. This is akin to the more modern idea of the "fight or flight" description of the human stress response (Auerbach & Gramling, 1998), a component of several theories of anxiety (e.g., Barlow, 1988). Freud (1964) also acknowledged the potential role of genetic contributions to individual differences in anxiety and its manifestations. Genetic variance in anxiety manifestation has received a considerable amount of research attention in recent years (e.g., Lesch et al., 1996) and holds promise for the future.

Other non-Freudian psychoanalytic theories of anxiety include the interpersonal theory of Sullivan (1953), in which the interpersonal field is emphasized as the primary context within which to describe personality development and functioning. According to Sullivan (1953), anxiety rises from a disturbance in the emotional connection between

mother and infant. Fairbairn (1952) and Guntrip (1968) present theories of anxiety based on object relations theory. Along these theoretical lines, the most intense anxiety has to do with ego breakdown and loss of the sense of self. Such theories are perhaps most evidently different from cognitive theories in terms of treatment, indicating relatively long-term therapy aimed at hypothesized underlying personality issues rather than cognitive errors and somatic symptoms. Although these theories are still espoused by some today, they have suffered from a relative dearth of empirical research. Despite the large number of empirical studies of anxiety in the past, most studies are "largely irrelevant to psychodynamic theories of anxiety" principally because controlled laboratory conditions often violate the terms of the theory (Eagle & Wolitzky, 1994).

Aside from psychoanalytic theories, others have emphasized various factors aside from catastrophic cognitions and focus on bodily sensations per se in the etiology of anxiety. The interaction model of personality (Endler & Magnusson, 1976) provides a theoretical perspective for examining the reciprocal influences of persons, situations, and responses, and proposes that a person's appraisal of the psychological meaning of a situation is an essential determining factor of behavior (e.g., anxiety and panic) (Endler & Edwards, 1994). Certainly, as we move along the spectrum of theories from those focusing on deeply-ingrained personality characteristics (e.g., Freud, 1959) to modern cognitive theories (e.g., Clark, 1988, Reiss, 1991), we see similarities and differences. One of the most important differences to highlight for the current work is the difference in treatment implications across theories. Cognitive theories, with their focus on cognitive "errors" and bodily symptoms, provide framework for brief interventions and possibly for prevention of anxiety. Anxiety sensitivity, one important component of

Reiss' (1991) expectancy theory, may be especially relevant for both treatment and prevention.

Anxiety Sensitivity

Bodily sensations do not invariably provoke panic, and in fact can provoke a wide variety of affective responses including pleasure (McNally, 1994). Reiss and McNally (1985) and Reiss (1991) proposed that preexisting beliefs regarding the harmfulness of symptoms determine whether someone will panic in response to bodily sensations. They assert that a trait known as anxiety sensitivity (AS) embodies such beliefs. The construct of AS refers to the extent to which an individual believes that autonomic arousal can have harmful consequences (Reiss & McNally, 1985). For example, individuals with high AS may believe that shortness of breath signals suffocation or that heart palpitations indicate a heart attack whereas those with low AS experience these sensations as unpleasant but nonthreatening.

Consistent with cognitive theories of anxiety, the AS conceptualization posits that cognitive misappraisal is critical for the generation of anxiety. However, the AS hypothesis is distinct from other psychological theories. For example, although AS is similar to Clark's (1986) "enduring tendency" to catastrophically misinterpret bodily sensations, a difference in conceptualizations is evident. Like Clark's panic patient, an individual with high AS is especially prone to catastrophic misinterpretation. However, people high in AS would not necessarily misinterpret sensations like rapid heartbeat as a heart attack. Rather, a high AS individual could become fearful because of a belief that rapid heartbeat can lead to heart attack. McNally (1994) explains that ". . . the anxiety-sensitivity hypothesis does not require that patients misconstrue anxiety as something

else (e.g., impending heart attack) for panic to be highly aversive" (p. 116). Anxiety sensitivity is believed to be a relatively stable belief system that may precede the development of panic attacks. Individual differences in AS are hypothesized to emerge from a variety of experiences that ultimately lead to the acquisition of beliefs about the potentially aversive consequences of arousal. Such experiences may include hearing others express fear of such sensations, receiving misinformation about the harmfulness of certain sensation, witnessing a catastrophic event such as the fatal heart attack of a loved one, and so forth. Thus, AS constitutes a disposition to developing anxiety and does not necessarily require the experience of anxiety or panic in its own development.

One of the major criticisms of AS is that it is not sufficiently distinct from trait anxiety (Lilienfeld, Turner, & Jacob, 1993). Overlap between trait anxiety and AS is expected according to models that describe AS as a lower order component of trait anxiety (Lilienfeld et al., 1993). However, trait anxiety and AS are conceptually distinct in that trait anxiety represents the general tendency to be fearful whereas AS represents the disposition to become fearful in the context of sympathetic arousal. Even though these constructs may be hierarchically related, psychometric (e.g., Taylor, Koch, & Crockett, 1991), nosological (Taylor, Koch, & McNally, 1992) and experimental studies (e.g., Telch & Harrington, 1992) indicate that AS and trait anxiety are distinct.

Taylor et al. (1991) combined the items of the Anxiety Sensitivity Index (ASI; Reiss, Peterson, Gursky, & McNally, 1986) and the Trait form of the State-Trait Anxiety Inventory (STAI-T; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) and subjected the compilation to factor analysis. Results revealed a two-factor solution in which the STAI-T items were responsible for almost all salient loadings on the first factor and the

ASI items were responsible for nearly all items on the second factor. Another study supported the nosological significance of the ASI. In this study, Taylor et al. (1992) found that mean STAI-T scores did not significantly differ among patients across the spectrum of anxiety disorders. However, ASI scores were significantly higher among patients with panic disorder than those with simple phobia, generalized anxiety disorder, social phobia, and obsessive-compulsive disorder. Finally, in an experimental study using a carbon dioxide challenge, Telch and Harrington (1992) found that ASI scores predicted challenge-induced panic even after statistically controlling for baseline levels of trait (STAI-T) anxiety.

Summary

These psychological models (i.e., Clark, 1986; Barlow, 1988, Reiss, 1991) are highly similar in terms of their hypothetical constructs and differ mainly with respect to more minor areas of focus (Rapee, 1993). In general, all of these models assume that panic is cued by somatic sensations and, therefore, somatic sensations should precede panic. One exception is Barlow's (1988) model in which it is not assumed that the perception of somatic sensations is always necessary for the precipitation of a panic attack. Sensations may be directly linked to panic via interoceptive conditioning. However, like Clark (1986), Barlow concedes that in many cases, panic is likely to be triggered by the perception of somatic sensations in patients with panic disorder. Rapee (1993) points out that Barlow's model becomes much more similar to other psychological models when the entire issue of panic disorder is considered (as opposed to only panic attacks), since Barlow acknowledges the role of sensations and interoceptive conditioning in panic disorder etiology. Another commonality among psychological models is the role

of psychological vulnerability. According to Clark (1986), certain individuals are vulnerable in that they have an enduring tendency to interpret bodily sensations in a negative fashion (i.e., elevated anxiety sensitivity). In Barlow's (1988) model, the first step in the panic disorder hierarchy is a biological vulnerability in which certain people are susceptible to developing panic attacks following stress ("false alarms"). However, Barlow also includes a psychological vulnerability component in his model where certain individuals are more likely to develop anxious apprehension and panic disorder. Anxiety sensitivity (Reiss, 1991) is proposed as one important component of this psychological vulnerability (Antony & Barlow, 1996a). It is important to note however, that although anxiety sensitivity fits in both Clark's and Barlow's model, anxiety sensitivity itself is not theoretically tied to interoceptive conditioning. It refers to beliefs about bodily sensations rather than conditioned fear responses to them.

Laboratory studies have yielded considerable support for psychological models of panic. Consistent with psychological hypotheses (most notably Clark, 1988), patients with panic disorder report that thoughts of danger typically accompany their panic attacks (Beck, Laude, & Bohnert, 1974; Hibbert, 1984; Ottavani & Beck, 1987), and such thoughts tend to occur after the detection of a bodily sensation (Hibbert, 1984; Ley, 1985a). Clark (1986) predicts that panic in response to a challenge procedure will occur only when the somatic sensations associated with the procedure are catastrophically interpreted. Indeed, such procedures produce similar sensations in both patients with panic disorder and nonclinical controls, but only patients consistently interpret the sensations as dangerous (e.g., Gaffney, Fenton, Lane, & Lake, 1988; Yeragani, Balon, & Pohl, 1989). In addition, patients with panic disorder who panic in response to challenge

procedures report thoughts of going crazy or losing control (i.e., catastrophic misinterpretations), while patients who do not panic when challenged report no such thoughts (Sanderson, 1988; Yeragani et al., 1989).

Evidence in support of the hypothesis that cognitive variables influence whether an individual panics during biological challenge is provided by studies that manipulate cognitive variables during challenges. It has been demonstrated that manipulation of expectancies can influence the degree of anxiety experienced during CO_2 inhalation (van den Hout & Griez, 1982), lactate infusion (van der Molen, van den Hout, Vroeman, Lousberg, & Griez, 1986), and hyperventilation (Salkovskis & Clark, 1989) in normal subjects. Rapee (1986) manipulated the pre CO_2 challenge instructions given to two groups of patients. One group was provided instructions that described the sensations induced by an inhalation of 50% CO_2 gas. All possible sensations were detailed, and their cause was attributed to the CO_2. A second group was provided with no explanation of the procedure. As predicted, the no explanation group reported significantly more catastrophic cognitions and significantly more panic in response to the inhalation than the detailed information group. Clark et al. (1992) tested the cognitive mediation hypothesis by manipulating pre-challenge instructions using a lactate challenge. Patients were randomized to one of two pre-infusion instructional sets, both of which emphasized that lactate is harmless and that the patient could stop the infusion at any time. In the experimental group however, the instructions also stressed that lactate is a normal substance, that it is normal to experience sensations during infusion, and that such sensations do not indicate bodily harm or danger. The experimental group was also encouraged to ask questions and were given answers emphasizing that strong sensations

are a normal effect of infusion. During the infusion, patients given the experimental instructions reported significantly less anxiety, less panic, and had smaller increases in heart rate. In both of these manipulations of interpretation (i.e., explanations for sensations), manipulation checks confirmed the validity of the different conditions (Rapee, 1986; Clark et al., 1992).

Margraf, Ehlers, Roth, Taylor, and Agras (1992) investigated cognitive mediation in patients with panic disorder during a hyperventilation challenge by manipulating expected affect. In this study, the procedure was described as either a "fast paced breathing task" or a "biological panic attack test." Consistent with the cognitive mediation hypothesis, patients in the "biological panic attack test" group had significantly higher expectations of anxiety prior to the challenge, rated the effects of the challenge as significantly more similar to natural panic attacks, and reported significantly more panic symptoms than those patients in the "fast paced breathing task" group.

Several researchers have argued that catastrophic misinterpretation *per se* is not a necessary ingredient for panic (e.g., Teasdale, 1988). In one study for example, Aronson, Whitaker-Asmitia, and Caraseti (1989) conducted a lactate challenge in which the presence of reassuring doctors prevented the patients from thinking catastrophic thoughts (according to self-report measures), but did not prevent them from panicking. Barlow's (1988) model explains this result with interoceptive conditioning, where Clark (1986) maintains that catastrophic misinterpretation may occur unconsciously. This condition also enables Clark to account for panic attacks that begin when the patient is asleep (McNally, 1991) and to disregard findings that patients with panic disorder insist that some attacks occur without catastrophic thoughts (Rachman, Lopatka, & Levitt, 1988).

Many argue that this aspect renders Clark's (1986) theory unfalsifiable. McNally (1994) points out that "If misinterpretations can be either conscious or unconscious, it is difficult to imagine what would count as evidence against the hypothesis that misinterpretations necessarily precede panics" (p. 115).

It is clear that these psychological theories of panic disorder are similar in many ways, the most notable differences involving the role of catastrophic thoughts (required, either consciously or unconsciously, by Clark) and the role of perceived control (i.e., Barlow, 1988). All theories are in agreement that certain individuals may be at a greater risk for developing panic disorder due to psychological vulnerabilities.

Risk Factors for Panic

Several risk factors for panic have been identified, including gender, age, and genetic makeup. Epidemiological data suggest that gender is a risk factor for panic (Robins et al., 1984; Katerndahl & Realini, 1993; Weissman & Merikangas, 1986). Females have approximately twice the lifetime risk for panic disorder than males after adjusting for race, marital status, and socioeconomic status. The median age of onset for panic disorder is 24, and young adults appear to be at the highest risk for the development of panic disorder (Eaton & Keyl, 1995; Barlow, 1988). Other studies have provided evidence that first degree relatives of panic disorder patients are at greater risk for the development of panic disorder including twin, family, and adoption studies (Torgerson, 1983; Noyes, Clancy, Crowe, Hoenk, & Slymen, 1978; Crowe, Noyes, Pauls, & Slymen, 1983). In addition, medical conditions such as a history of migraines appear to increase risk for the development of anxiety pathology (Breslau & Davis,1993). Reasons behind the link between medical conditions such as migraine and panic are unclear. This risk

may be related to an underlying pathophysiologic factor or, on the other hand, may be due to the unpleasant sensations associated with the condition. Individuals with uncomfortable medical conditions may be at increased risk for the development of heightened anxiety sensitivity which, in turn, mediates the development of panic. Schmidt and Telch (1997) found that the general perception of poor physical health among patients with panic disorder was significantly related to anxiety sensitivity both prior to treatment and six months following treatment. Finally, evaluation of candidate genes has begun to suggest specific that specific genes may be involved in the etiology of panic (e.g., Lesch et al., 1996).

In terms of psychological risk, several theories have proposed a variety of parameters that are implicated in the pathogenesis of anxiety and panic. For example, the experience of negative life events such as loss (e.g., death) has been associated with the development of panic (Faravelli & Pallanti, 1986). Two controlled studies found that patients with panic disorder experienced more negative life events during the year prior to panic than a matched group of nonclinical controls during the same period (Roy-Byrne, Geraci, and Uhde, 1986; Faravelli and Pallanti, 1989). Despite these findings, determining their exact etiological significance is difficult (McNally, 1984). These studies suffer from their retrospective nature, and it is possible that patients with panic disorder may be predisposed to recall negative and threatening life events (Barlow, 1988). Only a small number of psychological factors that have been examined in relation to anxiety and panic have been empirically established as risk factors. In recent years, converging evidence highlights anxiety sensitivity as one important factor.

Establishing Anxiety Sensitivity as a Risk Factor for Panic Disorder

One of the important and unique predictions of the anxiety sensitivity conceptualization is that anxiety sensitivity should act as a risk factor for the development of panic attacks as well as related anxiety disorders. Taken together, laboratory and prospective studies provide converging evidence for anxiety sensitivity as a risk factor in the development of anxiety pathology.

Experimental Studies

Challenge studies using nonclinical subjects with no history of spontaneous panic have demonstrated that AS is predictive of fearful responding to hyperventilation, caffeine, and 35% carbon dioxide inhalation (Donnell & McNally, 1989; Harrington, Schmidt, & Telch, 1996; Schmidt & Telch, 1994; Rapee & Medoro, 1994; Telch, Silverman, & Schmidt, 1996). For example, Schmidt and Telch (1994) investigated the singular and joint effects of AS and perceived safety of hypocapnia-induced bodily cues on nonclinical subjects' subjective and psychophysiological response to a 2 minute hyperventilation challenge. Subjects with no history of spontaneous panic were randomly assigned to one of two informational conditions (Safety Information versus No Safety Information). When anticipating hyperventilation, High AS-Safety Information subjects reported higher subjective anxiety compared to Low AS-Safety Information subjects. During hyperventilation, anxiety and safety information exerted independent effects on subjective responding. High AS subjects reported higher levels of subjective fear and physical symptoms compared to Low AS subjects; subjects who received safety information reported lower levels of anxiety and physical symptoms compared to those who did not receive safety information. High AS subjects' heightened subjective fear response persisted through the hyperventilation recovery period.

Telch, Silverman, and Schmidt (1996) assessed the effects of AS and perceived control on emotional responding to a caffeine challenge in 72 subjects with no history of panic disorder. Subjects high and low in AS were randomly assigned to either a perceived control (PC) or a no perceived control (NPC) instructional set. Compared to subjects low in AS, subjects high in AS displayed heightened emotional responding to the caffeine challenge. As predicted, high AS subjects in the NPC condition displayed significantly greater emotional responding compared to high AS subjects in the PC condition. In contrast, low AS subjects' emotional response to caffeine was not affected by the perceived control manipulation.

Treatment Outcome Studies

According to the anxiety sensitivity hypothesis, high levels of anxiety sensitivity should be associated with, and maintain, pathological anxiety among those already afflicted with an anxiety disorder. Likewise, anxiety sensitivity reductions associated with treatment should be associated with clinical improvement and may also predict long-term outcome (Schmidt, 1998). Several studies indicate that anxiety sensitivity is associated with changes in anxiety in clinical samples. Schmidt and Telch (1997) evaluated the relationship between changes in AS and recovery from panic disorder in 89 patients completing a 12 session cognitive behavioral therapy protocol or a 12-week delayed-treatment control. Mediation analyses based on composite recovery criteria (i.e., panic attacks, panic-related worry, phobic avoidance) indicate that changes in AS mediated the relationship between treatment and recovery. More specifically, AS was also found to mediate changes in anxiety symptoms and phobic avoidance. In a similar study, the level of AS at post-treatment as well as treatment-related changes in AS were

used to predict clinical status in 59 patients with panic disorder at 12-month follow-up. Both level of AS and change in AS were significantly related to anxiety symptoms, phobic avoidance, and overall disability at follow-up (Schmidt, 1997b).

Two reports have longitudinally evaluated anxiety sensitivity in clinical and subclinical populations (Bruce et al., 1995; Ehlers, 1995). Both studies suggest that elevated anxiety sensitivity is related to negative long-term outcome in terms of anxiety symptoms.

Ehlers (1995) found that high levels of AS predicted poorer outcomes in terms of maintenance, relapse, and frequency of symptoms among untreated patients and also predicted the frequency of untreated panic among untreated "infrequent" panic patients. After controlling for panic history, anxiety sensitivity levels predicted panic disorder status at follow up. At follow up, untreated "infrequent" panickers were more likely to experience continued panic attacks if they had high levels of anxiety sensitivity. In addition, even after controlling for the effects of previous panic attack frequency and other predictor variables anxiety sensitivity showed incremental validity in predicting the maintenance of panic disorder and panic attacks in the total sample (both clinical and subclinical patients).

Bruce et al. (1995) evaluated predictors of benzodiazepine discontinuation success among patients with panic disorder completing either a flexible drug taper schedule plus standard medical management or the same taper schedule with CBT. Ninety percent of patients receiving CBT compared to only forty percent of patients receiving standard medical management were still medication free at follow-up. Several predictor variables were evaluated, including severity of withdrawal symptoms,

pretreatment benzodiazepine dose, duration of previous medication treatment, and level of phobic avoidance, in addition to ASI scores. Only changes in ASI scores from baseline were predictive of medication status at follow-up. This finding indicates not only that reductions in anxiety sensitivity were associated with receiving CBT, but also that reductions in anxiety sensitivity were associated with benzodiazepine discontinuation. Thus, posttreatment assessments of change in anxiety sensitivity may be extremely important in predicting adverse outcomes of benzodiazepine taper, especially among patients who have not received CBT (Schmidt, 1998). Perhaps more important to the study of anxiety sensitivity, this finding also provides evidence that anxiety sensitivity can be lowered in clinical populations. However, Bruce et al. (1995) did not report the clinical status of participants, making it difficult to make conclusions regarding the effect of changes in anxiety sensitivity on clinical condition other than the use of medication (Schmidt, 1998).

Over the past few years, our lab has collected anxiety sensitivity data (both change in ASI score from pretreatment levels and absolute ASI scores at posttreatment) as part of treatment outcome studies of panic disorder. Ongoing treatment has been conducted using a protocol of twelve sessions of CBT (e.g., Schmidt, Staab, Trakowski, & Sammons, 1997). All patients treated were diagnosed with a principal DSM-IV diagnosis of panic disorder, and were reassessed at posttreatment and three months following treatment. An analysis of 37 patients who underwent this treatment protocol indicated an average pretreatment ASI score of 29.3, an average posttreatment score of 12.1, and an average follow-up score of 11.1. The average level of reduction based on a pre-post change score was 17 points, suggesting that the treatment exerted a substantial

effect on anxiety sensitivity. Anxiety sensitivity scores at posttreatment are lower than might be expected in nonclinical populations (Taylor et al., 1992; Telch, Shermis, & Lucas, 1989) suggesting especially effective treatment in this case. Follow-up scores in comparison to posttreatment scores are indicative of good maintenance of these substantial anxiety sensitivity reductions (Schmidt, 1998).

Prospective Studies with Nonclinical Populations

While longitudinal studies of clinical populations allow for the examination of the role of anxiety sensitivity in the long term course of anxiety pathology, prospective studies of anxiety sensitivity in nonclinical samples permit the evaluation of the initial incidence of anxiety pathology. Such studies are important in evaluating the effects of anxiety sensitivity because in nonclinical and symptom-free individuals with no history of anxiety disorder, anxiety sensitivity cannot be attributed to preexisting anxiety pathology (Schmidt, 1998). Aside from studies included in the current paper, only two previous studies evaluated the relationship between anxiety sensitivity and the development of anxiety in nonclinical samples (Ehlers, 1995; Maller & Reiss, 1992). Both suggested that individuals with elevated anxiety sensitivity are at increased risk for the development of anxiety pathology.

Maller and Reiss (1992) conducted a three year follow-up (based on structured diagnostic interviews as well as self-report measures) using a nonclinical sample of college students originally assessed with the ASI. Scores on the ASI were predictive of the frequency and intensity of panic attacks during the follow-up period. Those with high ASI scores (mean ASI score of 33) were five times more likely than those with low anxiety sensitivity (ASI mean of 11) to have developed an anxiety disorder during the

follow-up period. At follow-up, nearly half of those in the high anxiety sensitivity group received a diagnosis of an anxiety disorder compared to only about one tenth of those in the low anxiety sensitivity group. In addition to diagnostic status, anxiety sensitivity was also associated with incidence of panic attacks and maintenance of anxiety pathology. Regarding the panic attack incidence, Four subjects (three of whom were in the high anxiety sensitivity group) experienced spontaneous panic attacks for the first time during the follow-up interval. Regarding the maintenance of anxiety pathology, Maller and Reiss (1992) report that of 18 subjects experiencing panic attacks during the follow-up period, only four were reporting panic for the first time. Eleven of the 18 subjects reporting panic were in the high anxiety sensitivity group. In contrast, 25 of the 30 subjects who did not experience panic had low ASI scores. Thus, most individuals with high anxiety sensitivity and a history of panic continued to experience panic attacks during the follow-up period. High anxiety sensitivity was also associated with greater frequency and intensity of panic attacks.

Although this initial study provides evidence of a link between anxiety sensitivity and the development of panic attacks, the relatively small sample size and uncertainty about preexisting psychiatric conditions precludes definitive conclusions about the role of anxiety in the development of anxiety pathology (Schmidt, 1998).

As noted above, Ehlers (1995) conducted a one year follow-up study of panic attacks and other anxiety-related symptoms in a sample consisting of patients with panic disorder, patients with panic disorder currently in remission, infrequent (subclinical) panickers, patients with simple phobias, and nonclinical controls with no history of spontaneous panic attacks or psychiatric disorders. Anxiety sensitivity was a significant

predictor of the first reported occurrence of spontaneous panic. Five subjects reported their first spontaneous panic attack during the follow-up period, and these individuals had significantly higher levels of anxiety sensitivity than those who did not report panic. Aside from a small sample size, this study represented a methodological improvement over the Maller and Reiss (1992) study because psychiatric diagnoses and trait anxiety were determined at the initial assessment. Anxiety sensitivity, but not trait anxiety, predicted the occurrence of new panic attacks during the follow-up.

Anxiety Sensitivity, Panic Disorder, and Other Risks

Cardiovascular Disease

Patients with panic disorder appear to be at increased risk for cardiovascular disease (Coryell, Noyes, & Clancy, 1982; Coryell, Noyes, & House, 1986; Weissman et al., 1990). Coryell and colleagues found approximately a twofold increase in cardiovascular mortality in patients with panic disorder compared to the general population (Coryell, Noyes, & Clancy, 1982; Coryell, Noyes, & House, 1986). Weissman et al.'s (1990) assessment of cardiovascular problems from the New Haven portion of the ECA survey found that a lifetime diagnosis of panic disorder was associated with higher risks for hypertension, myocardial infarction, and stroke relative to those with no history of psychiatric disorders. Evidence for increased mortality and CHD among patients with panic disorder has led to speculation regarding a variety of pathophysiological mechanisms including mitral valve prolapse (MVP), decreased heart rate variability, and panic-related hyperventilation-precipitated coronary spasm (Goldberg, 1988). However, these factors best account for cases of sudden death among patients with panic disorder and do not readily explain CHD-related findings.

Decreased exercise participation is one other distal factor that may be associated with increased risk for CHD in patients with panic disorder. Although research is limited, several studies have examined the relationship between panic disorder, cardiovascular fitness, and exercise (Cameron & Hudson, 1986; Taylor et al., 1987; Gaffney, Fenton, Lane, & Lake, 1988; Stein et al., 1992). For example, Cameron and Hudson (1986) found that many patients with panic disorder were characterized as "exercise sensitive" (i.e., reporting severe anxiety to exercise).

Cognitive variables such as anxiety sensitivity may help elucidate the link between panic disorder and CHD. Patients with panic disorder generally exhibit high levels of anxiety sensitivity , and for some, high anxiety sensitivity scores may be due to cardiac-specific fears (e.g., "it scares me when my heart beats rapidly) (Taylor, Koch, McNally, & Crockett, 1992). Thus, cardiopulmonary fears (i.e., high anxiety sensitivity) may be responsible for exercise intolerance and physical inactivity due to catastrophic misinterpretation regarding the symptoms typically produced during exertion.

Pain

Although the vast majority of research concerning anxiety sensitivity has centered on the anxiety disorders, researchers have recently begun to examine the role of AS in other conditions including pain. This research may have been sparked by the finding that there is a high degree of co-morbidity between chronic pain and anxiety disorders (Asmundson, Jacobson, Allerdings, & Norton, 1996). Individuals with chronic pain tend to be more anxious that non-clinical populations (Craig, 1994) and patients with panic disorder patients frequently report significant and persistent pain (Schmidt & Telch, 1997).

The work of Asmundson and colleagues (Asmundson et al., 1996; Asmundson, Kuperos, & Norton, 1997; Asmundson & Taylor, 1996) suggests that AS may play a role in the onset and or maintenance of chronic pain. Asmundson suggests that since AS marks an individual's general propensity to develop fears, it may amplify a specific tendency to develop fear of pain. Fear of pain, in turn, increases pain related escape/avoidance behaviors which have been proposed as important mechanisms of pain maintenance (Fordyce, 1976; Lethem, Slade, Troup, & Bentley, 1983; Philips, 1987). More specifically, negative expectancies regarding the harmfulness of pain are amplified by AS, causing avoidance behaviors, and these avoidance behaviors lead to deconditioning (e.g., muscular atrophy, reduction in activity, and weight gain). Deconditioning leads to increased pain which heightens avoidance and exacerbates negative expectancies regarding pain (Asmundson, Norton, & Norton in press). Thus, AS may be a key factor in the maintenance of pain and disability through its action of enhancing the reciprocal relationship between fear and avoidance.

Depression

Recently, several reports have found an association between anxiety sensitivity and depression (Otto, Pollack, Fava, Uccello, & Rosenbaum, 1995; Taylor, Koch, Woody, & McLean, 1996). Otto et al. (1995) reported increased ASI scores in patients with major depression. Scores for depressed participants were somewhat lower than patients with panic disorder but were comparable to other anxiety disorders such as generalized anxiety disorder, social phobia, and specific phobia. Taylor et al. (1996) also found ASI scores to be elevated in patients with major depression. In addition, patients

with panic disorder with co-occurring major depression showed higher scores compared to those panic disorder patients without depression.

The Current Studies

The present report includes a series of five reports investigating the role of anxiety sensitivity in a number of areas. As opposed many studies reviewed above, only one of these is a laboratory based study. In addition to investigating the role of anxiety sensitivity, the concept of psychological vulnerability factors in general is explored, and other possible risk factors such as discomfort intolerance are examined.

The first three reports presented here stem from a large sample of data collected at the U.S. Air Force Academy (USAFA) during Basic Cadet Training (BCT) in the summer of 1995. The Air Force Academy was chosen as a data collection site for several reasons. We initially considered the Academy for the practical reason that it would provide a large sample of research participants previously inaccessible to our lab. We also realized that other benefits were specific to USAFA. For example, the context of basic training is ideal for evaluating individuals under a great amount of stress. Our research, in part, attests to the diathesis-stress model of psychopathology and such an environment would provide an ideal test of psychological diatheses. Also, it was hoped that because Cadets are easily followed in comparison to other populations (they are geographically isolated and divided into easily identifiable subgroups, or squadrons) longer term follow-up might be possible. Over one thousand cadets were evaluated over the course of their first five weeks of training. Self-report instruments were administered as part of other standard data collection batteries that all basic trainees complete. Our self-report battery was quite extensive, and included measures of demographic data,

psychological history, numerous hypothesized psychological vulnerability factors such as anxiety sensitivity, and psychological distress (e.g., anxiety and depression symptoms). Data were collected once at the beginning of basic training and again during the last week of basic training.

Report 1: Schmidt, N.B., Lerew, D.R., & Jackson, R.J. (1997). The role of anxiety sensitivity in the pathogenesis of panic: Prospective evaluation of spontaneous panic attacks during acute stress. Journal of Abnormal Psychology, 106, 355-364.

This report, the first to be published in the series, focuses specifically on anxiety sensitivity and its role as a vulnerability factor in the pathogenesis of anxiety pathology. Panic attacks are the main outcome variable discussed in addition to anxiety and depression symptoms. Functional impairment is discussed briefly, but a focus is maintained on the prospective test of whether anxiety sensitivity acts as a premorbid risk factor for anxiety pathology.

Report 2: Schmidt, N.B., & Lerew, D.R. (in press). Prospective evaluation of psychological risk factors as predictors of functional impairment during acute stress. Journal of Occupational Rehabilitation.

This report, somewhat unique in its target audience (i.e., occupational rehabilitation professionals), focuses on the relationship between psychological risk factors and the development of impairment and disability regardless of mediating factors such as anxiety and depression. In addition to anxiety sensitivity, this report explores the role of two other vulnerability factors--body vigilance and discomfort intolerance. Impairment is measured in terms of peer

relations, supervisory relations, physical health, and overall performance. Disability was evaluated in terms of peer counseling visits, counseling center visits, clinic visits, and sick days. Gender is also evaluated as a potential moderator variable.

Report 3: Lerew, D.R., Schmidt, N.B., & Jackson, R.J. (in press). Evaluation of psychological risk factors: Prospective prediction of psychopathology during basic training. Military Medicine.

This report, with an eye toward the military training and military adaptability aspects of psychological problems, addresses reassurance-seeking and perceived vulnerability in addition to anxiety sensitivity. Results are discussed in terms of the diathesis-stress model, the adaptability of military trainees, and the nature of military training.

The remaining two studies presented here take a somewhat different approach, reflecting a change in focus away from the relationship between psychological vulnerability factors and psychopathology per se and toward the theoretical place for anxiety sensitivity in a broader sense (i.e., Schmidt, Lerew, & Joiner, 1998) and to other more distal effects of elevated anxiety sensitivity (i.e., Schmidt, Lerew, Santiago, Trakowski, & Staab, under review).

Report 4: Schmidt, N.B., Lerew, D.R., & Joiner, T.E., Jr. (1998). Anxiety sensitivity and the pathogenesis of anxiety and depression: Evidence for symptom specificity. Behavior Research and Therapy, 36, 165-177.

Schmidt, Lerew, and Joiner (1998) evaluate anxiety sensitivity in light of evidence suggesting an association between this variable and depression. If

anxiety sensitivity is a vulnerability factor for depression in addition to anxiety, it loses its conceptual specificity as a risk factor for anxiety, and could be considered instead as a general risk factor for psychopathology. This report utilizes a clinical sample in addition to the sample collected at USAFA in order to examine the relationship between anxiety sensitivity and depression.

Report 5: Schmidt, N.B., Lerew, D.R., Santiago, H., Trakowski, J.H., & Staab, J.P. (under review). American Journal of Psychiatry.

The final manuscript included in the current work is considerably different from the previous reports. This study evaluates the potential relationship between anxiety sensitivity, panic disorder, cardiovascular fitness, and exercise. It is hypothesized that anxiety sensitivity may act as a cognitive mediator such that individuals high in anxiety sensitivity may avoid exercise. In addition, heart rate perception is manipulated to determine its effect on fitness estimation. Results suggest that patients with panic disorder exhibit poorer cardiovascular fitness than normal controls.

Running Head: ANXIETY SENSITIVITY AND PANIC

The Role of Anxiety Sensitivity in the Pathogenesis of Panic: Prospective Evaluation of Spontaneous Panic Attacks During Acute Stress

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Abstract

Expectancy theory posits that anxiety sensitivity may serve as a premorbid risk factor for the development of anxiety pathology (Reiss, 1991). The principal aim of the present study was to determine whether anxiety sensitivity acts as a specific vulnerability factor in the pathogenesis of anxiety pathology. A large nonclinical sample of young adults (N = 1401) was prospectively followed over a five week highly stressful period of time (i.e., military basic training). Anxiety sensitivity was found to predict the development of spontaneous panic attacks after controlling for a history of panic attacks and trait anxiety. Approximately 20% of those scoring in the upper decile on the Anxiety Sensitivity Index (Peterson & Reiss, 1987) experienced a panic attack during the five week follow-up period compared to only 6% for the remainder of the sample. Anxiety sensitivity also predicted anxiety symptomatology, functional impairment created by anxiety, and disability. These data provide strong evidence for anxiety sensitivity as a risk factor in the development of panic attacks and other anxiety symptoms.

The Role of Anxiety Sensitivity in the Pathogenesis of Panic: Prospective Evaluation of Spontaneous Panic Attacks During Acute Stress

Panic attacks are discrete and intense periods of autonomic arousal accompanied by fear. Spontaneous panic, as opposed to situationally bound or situationally disposed panic, is unexpected and not readily associated with a triggering stimulus. Panic attacks, in particular spontaneous panic, are the central and defining feature of panic disorder, but panic attacks are not limited to those experiencing panic disorder or agoraphobia. Panic is common to all the anxiety disorders (Barlow et al., 1985); hence its separation from panic disorder in the most recent revision of the DSM (DSM-IV; American Psychiatric Association, 1994). In fact, surveys of nonclinical samples suggest that a substantial number of adults experience spontaneous panic attacks (Norton, Dorward, & Cox, 1986; Norton, Harrison, Hauch, & Rhodes, 1985). One study of over 2,000 college students indicated a lifetime prevalence of approximately 12% for spontaneous panic (Telch, Lucas, & Nelson, 1989).

"Fear of fear" has played an important role in the conceptualization and research on both panic attacks and panic disorder. Goldstein and Chambless (1978) suggested that panic disorder patients learn to fear symptoms of anxiety through interoceptive classical conditioning of internal physical sensations. Internal bodily cues become a conditioned stimulus for the conditioned response of anxiety and panic. According to Goldstein and Chambless, fear of fear develops as a consequence of having panic attacks. Fear of fear has also been described within cognitive theories of panic (Beck & Emery, 1985; Clark, 1986) which suggest that panic attacks arise from the catastrophic misinterpretation of benign bodily perturbations. Misinterpretation of bodily cues leads to a vicious cycle in which

faulty interpretation leads to more anxiety as a fearful response to arousal increases the very symptoms that constitute the focus of apprehension. This process may ultimately spiral into full-blown panic as fear feeds upon itself (see McNally, 1990).

The construct of anxiety sensitivity is closely linked with fear of fear (Reiss & McNally, 1985). Anxiety sensitivity refers to the extent to which an individual believes that autonomic arousal can have harmful consequences. For example, individuals with high anxiety sensitivity may believe that shortness of breath signals suffocation or that heart palpitations indicate a heart attack whereas those with low anxiety sensitivity experience these sensations as unpleasant but nonthreatening.

Consistent with cognitive theories of anxiety, the anxiety sensitivity conceptualization posits that cognitive misappraisal is critical for the generation of anxiety. However, anxiety sensitivity is distinguished from other cognitive conceptualizations because anxiety sensitivity is believed to be a stable trait-like characteristic that may precede the development of panic attacks. Individual differences in anxiety sensitivity are hypothesized to emerge from a variety of experiences that ultimately lead to the acquisition of beliefs about the potentially aversive consequences of arousal. Such experiences may include hearing others express fear of such sensations, receiving misinformation about the harmfulness of certain sensations, witnessing a catastrophic event such as the fatal heart attack of a loved one, and so forth. Thus, anxiety sensitivity constitutes a disposition to developing anxiety and does not require the experience of anxiety or panic in its own development.

The anxiety sensitivity conceptualization of fear is not without controversy. One of the major criticisms leveled against this conceptualization is that prior research has not clearly demonstrated that anxiety sensitivity is distinct from trait anxiety (Lilienfeld, Turner,

& Jacob, 1993). Although there are conceptual distinctions between anxiety sensitivity (i.e., disposition to become fearful in the context of sympathetic arousal) and trait anxiety (i.e., general tendency to be fearful across a broad array of stimuli), overlap between these constructs is expected according to models that describe anxiety sensitivity as a lower order component of trait anxiety (Lilienfeld et al., 1993). Even though these constructs may be hierarchically related, psychometric, nosological, and experimental studies indicate that anxiety sensitivity and trait anxiety are distinct (see McNally, 1996 for a review). For example, McNally (1989) reported that anxiety sensitivity was a better predictor of fearful responding to hyperventilation than trait anxiety. Yet, as Lilienfeld et al. highlight, the discrimination of anxiety sensitivity from trait anxiety deserves further evaluation.

Although anxiety sensitivity is elevated in panic disorder as well as other anxiety disorders (Taylor, Koch, & McNally, 1992), and anxiety sensitivity decreases with remission of panic disorder symptomatology (Telch et al., 1993), these studies only confirm that anxiety sensitivity is a concomitant of panic disorder. One of the important predictions of anxiety sensitivity conceptualizations is that anxiety sensitivity should act as a risk factor for the development of panic attacks as well as related conditions such as panic disorder. Data supporting this prediction are limited. Some evidence suggests that high levels of anxiety sensitivity can precede the development of panic attacks. Donnell and McNally (1990) found a large number of college students scoring high on the Anxiety Sensitivity Index (ASI), a measure of anxiety sensitivity, with no history of spontaneous panic attacks. High anxiety sensitivity, independent of a history of panic, has also been found to predict anxious responding to biological challenge (i.e., exposure to substances or procedures that result in physiological changes). Challenge studies using nonclinical subjects with no history of

spontaneous panic have demonstrated that anxiety sensitivity is predictive of fearful responding to hyperventilation, caffeine, and 35% carbon dioxide inhalation (Donnell & McNally, 1990; Harrington, Schmidt, & Telch, 1996; Schmidt & Telch, 1994; Rapee & Medoro, 1994; Telch, Silverman, & Schmidt, 1996). In addition, a causal modeling study using nonclinical subjects found that anxiety sensitivity was associated with agoraphobic fears and avoidance (Taylor and Rachman, 1992). Because Taylor and Rachman did not assess panic attacks, it is unclear what role panic may have played in these relationships.

Only one study has prospectively evaluated a nonclinical sample with no history of panic to provide a more direct test of whether anxiety sensitivity acts as a risk factor for the subsequent development of panic. Maller and Reiss (1992) conducted a three year follow-up study using a nonclinical sample of college students originally assessed with the ASI. Scores on the ASI were predictive of the frequency and intensity of panic attacks during the follow-up period. Those with high ASI scores were five times more likely to have developed an anxiety disorder during the follow-up period. These findings offer the best evidence that anxiety sensitivity may act as a risk factor for anxiety pathology. However, the small sample size (data for only 48 subjects were obtained at follow-up) precludes definitive conclusions and highlights the need for additional prospective evaluations.

The present study prospectively evaluated whether anxiety sensitivity is associated with the development of anxiety pathology in a large sample of nonclinical subjects. One of the unique aspects of the present study is that the participants were followed over a relatively brief (i.e., five weeks) but highly stressful period of time. The United States Air Force Academy provided an opportunity to evaluate incoming first year students during their initial five weeks of training at the academy. This period, called Basic Cadet Training (BCT),

consists of highly regimented training in the context of fairly extreme psychosocial (e.g., isolation from friends and family, constant monitoring and evaluation of behavior) as well as physical (e.g., intense exercise, limited sleep) stressors. These BCT specific factors, in conjunction with the general stressors associated with entering college (e.g., first period away from home for many of these young adults), make BCT an ideal environment for engendering anxiety.

The present study evaluated over one thousand cadets during the first and last weeks of BCT to determine whether anxiety sensitivity predicted the development of anxiety pathology. It was hypothesized that anxiety sensitivity would act as a cognitive diathesis that would place individuals at risk for the development of psychopathology in the context of the substantial stressors associated with BCT. More specifically, it was hypothesized that anxiety sensitivity, independent of a history of panic attacks as well as trait anxiety, would predict the development of spontaneous panic and other anxiety symptoms as well as impairment and disability.

Method

Participants and Procedure

Participants included 1401 first year undergraduate students (i.e., cadets) from the United States Air Force Academy (USAFA). General screening criteria for admission to USAFA include: (1) a congressional or presidential nomination, (2) passing a Department of Defense medical screen, (3) passing a criminal record review, (4) between the ages of 17 and 22, and (5) single with no dependents. From the total pool, 132 cadets were excluded because of incomplete data from the initial (Time 1) assessment. An additional 97 cadets were excluded based on a screening questionnaire that included all items from the MMPI-2

Lie scale (Butcher, 1990). Those scoring greater than 5 on the MMPI-2 Lie scale were excluded to control for defensive responding. Excluded cadets did not differ from the remaining sample in terms of demographics and clinical variables (ps > .05). The remaining participants (N = 1172) were largely male (84%) and Caucasian (84%) with a mean age of 18.0 (SD = 0.9).

The educational requirements of USAFA include military training that begins upon arrival with Basic Cadet Training (BCT). The BCT period consists of five weeks of training divided into two phases. The first phase is characterized by administrative tasks including placement tests for the academic classes that begin after BCT as well as military indoctrination (e.g., marching, study of military history). The second phase of BCT focuses on physical training. The physical surroundings of the second phase are more extreme as cadets move from the dormitory to tents in the training field. In this context, cadets participate in highly demanding physical activities (e.g., long runs, obstacle courses). In general, BCT is designed to continuously expose cadets to a variety of unpredictable and uncontrollable physical and mental stressors. Cadets are not given schedules and have no access to clocks or watches. They cannot predict whether their next activity will be an academic evaluation, a military exercise, or a five mile run. New stressors are continually introduced to ensure that each cadet is overtaxed.

Data for the present study were gathered during a group administration of measures to the entire class of cadets during the first few days of BCT (Time 1) and five weeks later at the end of BCT (Time 2). Participants were told that the study was evaluating the impact of BCT on physical and emotional functioning. Prior to the administration of measures, cadets were assured that USAFA would not have access to information collected. Furthermore,

code numbers were utilized on all forms to ensure anonymity. Written informed consent was also obtained. The Time 1 assessment battery consisted of measures of anxiety sensitivity, history of spontaneous panic, trait anxiety, anxiety symptoms, depression and hopelessness, as well as indices tapping the functional impairment created by anxiety. The Time 2 assessment battery was identical to the Time 1 battery but also included measures of spontaneous panic, panic frequency, and panic-related worry during BCT as well as indices of physical and mental disability. Data on voluntary and involuntary separations (i.e., cadets choosing or forced to leave) from USAFA were obtained from the USAFA Counseling Center.

Measures

Anxiety Sensitivity Index (ASI). The ASI is a 16-item questionnaire that measures fear of arousal symptoms (Peterson & Reiss, 1987). Each item assesses concern about the possible negative consequences of anxiety symptoms. The ASI has demonstrated adequate internal consistency (Telch, Shermis, and Lucas, 1989) and test-retest reliability (Maller & Reiss, 1992). Moreover, the ASI appears to tap fear of anxiety symptoms as opposed to state or trait anxiety (see McNally, 1994).

Beck Anxiety Inventory (BAI). The BAI is a 21-item measure of anxiety symptoms (Beck, Epstein, Brown & Steer, 1988). Each item assesses the degree to which physical or cognitive symptoms of anxiety have affected the individual during the past week. The BAI has been shown to be a reliable and valid measure of anxiety in a variety of studies using both clinical (coefficient alpha = .92) and nonclinical (coefficient alpha = .91) samples (Beck et al., 1988; Borden, Peterson, Jackson, 1991).

Beck Depression Inventory (BDI). Level of depressive symptoms experienced during the past week was assessed by the 21-item BDI. The BDI is a reliable and well-validated measure of depressive symptomatology (see Beck, Steer, & Garbin, 1988 for a review). Beck, Steer et al. (1988) reported high internal consistency (mean coefficient alpha = .81) for nonclinical populations.

Beck Hopelessness Scale (BHS). Level of hopelessness was assessed by the 20-item BHS. The BHS is a reliable and validated measure of pessimism about the future (Beck, Weissman, Lester, & Trexler, 1974). Beck, Steer, Sanderson, and Skeie (1991) reported high internal consistency for clinical and nonclinical populations (average coefficient alphas in .80s).

Panic History Form. The Panic History Form is a 4-item screening instrument for assessing history of spontaneous panic attacks (i.e., Have you ever experienced a sudden and intense surge of fear or anxiety (e.g., a panic attack) in a situation for no apparent reason?), panic attack frequency (i.e., Have you ever experienced four or more panic attacks in the period of one month?), panic-related worry (i.e., Have you ever had a panic attack and subsequently worried about having another for one month or more?), and a general history of psychological and psychiatric treatment (i.e., Have you ever seen a psychologist, psychiatrist, or other mental health professional for treatment?). Expected (i.e., situationally bound or disposed) panic attacks were not assessed. This instrument reliably detects a history of spontaneous panic in nonclinical samples (Schmidt & Telch, 1994; Telch, Silverman, & Schmidt, 1996). Data from a sample of patients presenting for evaluation and treatment indicate that substantial agreement between endorsement of panic attacks and structured interview diagnosis of panic attacks (r = .64). Data from nonclinical samples also indicates

substantial agreement between self-reported and interview diagnosis of panic attacks (r = .58). Compared to structured diagnostic interview, Panic History Form rates of false positive diagnoses of spontaneous panic among nonclinical samples are 45% for participants endorsing at least one spontaneous panic attack, 30% for participants endorsing four panic attacks in a one month period, and 30% for participants endorsing one month or more of panic-related worry. The time frame of this instrument was modified for the Time 2 assessment to cover only the five week BCT period. Participants also rated the number of spontaneous panic attacks experienced during BCT.

State-Trait Anxiety Inventory (STAI). The STAI is composed of two 20-item scales designed to assess state and trait anxiety. Both scales of the STAI have adequate psychometric properties (Knight, Waal-Manning, & Spears, 1983). Only the trait scale, which measures the general tendency to react with anxiety to a wide range of stimuli, was utilized in the present study.

Impairment, Disability and Attrition. In order to assess the impact of anxiety pathology, a self-report questionnaire was constructed to index impairment and disability. Impairment was assessed using four Likert-format questions (range: 0 = Not at All to 10 = Extremely) rating the degree to which symptoms of anxiety negatively affected: (a) peer relations, (b) supervisory relations, (c) physical health and well-being, and (d) overall performance. The Impairment index showed adequate internal consistency in the present sample (□ coefficient = .78). Disability was evaluated using four questions that assessed the frequency of: (a) peer counseling, (b) counseling center visits, (c) clinic visits for physical illness, and (d) sick call (i.e., absent from activity due to illness). Attrition was assessed from

data obtained from the USAFA Counseling Center which processes all cadets leaving the Academy.

Results

History of Spontaneous Panic and Clinical Characteristics at Time 1

Evaluation of the panic history question indicated that 14% of participants reported at least one "out of the blue" panic attack at some prior point in their lives. Only 4% reported four or more panic attacks in any one month period. Similarly, only 4% reported one month of worry about panic or significant change in behavior subsequent to having a panic attack. In addition, 4% reported a lifetime history of seeing a mental health professional.

Overall, the sample reported levels of symptomatology consistent with normative data for nonclinical undergraduate populations except for slight elevations of anxiety symptomatology (BAI: $\underline{M}$ = 18.0, $\underline{SD}$ = 10.6). Scores on the other self-report measures suggested that trait anxiety (STAI: $\underline{M}$ = 42.5, $\underline{SD}$ = 5.3) depression (BDI: $\underline{M}$ = 9.7, $\underline{SD}$ = 7.2), and hopelessness (BHS: $\underline{M}$ = 2.9, $\underline{SD}$ = 2.9) were generally within the normal range of functioning. Levels of anxiety sensitivity were low (ASI: $\underline{M}$ = 4.0, $\underline{SD}$ = 2.9) in comparison with other nonclinical college samples (see Telch, Lucas, & Nelson, 1989). Correlations among the major clinical indices are provided in Table 1. The general pattern of correlations indicates that anxiety sensitivity is significantly associated with symptomatology and impairment at both Time 1 and Time 2 ($\underline{ps} < .01$) whereas trait anxiety is not significantly associated with most outcome measures at either assessment period ($\underline{ps} > .05$).

Table 1

Intercorrelations, Means and Standard Deviations among Major Indices at Time 1 (below Diagonal) and Time 2 (above Diagonal)

	1	2	3	4	5	6	M	SD
1. ASI	(.65)	.05	.31*	.19*	.08*	.11*	3.8	5.4
2. STAI	.12*	(.68)	-.02	-.06	-.05	-.02	42.7	5.3
3. BAI	.30*	.09*	(.53)	.53*	.25*	.44*	9.6	7.3
4. BDI	.24*	.07	.60*	(.55)	.55*	.43*	6.4	6.2
5. BHS	.10*	.01	.32*	.58*	(.48)	.25*	2.2	2.3
6. Impairment	.21*	.01	.51*	.53*	.34*	(.34)	9.6	7.9
M		4.0	42.5	18.0	9.7	2.9	15.9	
SD		2.9	5.3	10.6	7.2	2.9	8.0	
α coefficient	.66	.72	.91	.86	.81	.78		

Note. Correlations among Time 1 measures appear below the diagonal and correlations among Time 2 measures appear above the diagonal. Correlations between scores on each measure at Time 1 and Time 2 appear in the diagonal.

1 - ASI = Anxiety Sensitivity Index; 2 - STAI = Spielberger Trait Anxiety Scale; 3 - BAI = Beck Anxiety Inventory; 4 - BDI = Beck Depression Inventory; 5 - BHS = Beck Hopelessness Scale; 6 - Impairment = Total Score from Impairment Scale.

*$p < .01$.

$n = 472$.

Spontaneous Panic Attacks during BCT

During the five week basic training period, approximately 6% (n = 74) of the sample reported experiencing at least one spontaneous panic attack. Of those reporting panic, the majority (62%) experienced only one panic attack with 16% having two panic attacks and 22% having three or more panic attacks. In addition, 28% reported worry about additional attacks or the consequences of having panic. Following the BCT period, those experiencing a panic attack, compared to cadets reporting no panic, evidenced significantly greater impairment in functioning and higher levels of psychopathology as indexed by the BAI, BDI and BHS (see Table 2).

Anxiety Sensitivity Predicting Spontaneous Panic

Logistic regression was used to determine whether Time 1 ASI scores predicted the development of spontaneous panic during BCT (see Table 3). As a first step, demographic variables (i.e., age, gender, race) were separately used to predict the development of panic. None of these analyses were significant (c^2 (1, N = 1117) = 0.17 (age), 0.88 (gender), (4, N = 1110) = 2.13 (race), ps > .05) and, accordingly, demographic variables were not entered as covariates in later analyses.

As predicted, the ASI was significantly associated with the development of spontaneous panic (c^2 (1, N = 1123) = 13.8, p < .001, r = .16). Although ASI scores for those experiencing panic (M = 7.5, SD = 3.6) were elevated relative to the remaining sample (M = 3.8, SD = 2.9), these scores are considerably lower than the average scores reported for

Table 2

Level of Impairment and Psychopathology for Cadets Experiencing Spontaneous Panic during Basic Cadet Training

	Cadet Group		
Time 2 Variable	Reporting Panic	No Panic	F
Overall Impairment			
M	14.3	9.3	34.1
SD	7.7	7.8	
n	74	1036	
Anxiety Symptoms (BAI)			
M	14.9	9.2	42.2
SD	8.1	7.1	
n	74	995	
Depression Symptoms (BDI)			
M	10.7	6.1	39.2
SD	7.9	5.9	
n	74	1008	
Hopelessness (BHS)			
M	3.3	2.1	18.0
SD	2.8	2.2	
n	74	995	

Note. Groups differ significantly on all measures (ps < .0001). BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory, BHS = Beck Hopelessness Scale.

nonclinical samples (Taylor et al., 1992). When the sample is divided into quartiles based on ASI scores, approximately 13% of those in the highest quartile experienced a panic attack compared to less than 3% for those in the lowest quartile (see Figure 1). Those in the highest quartile ASI group showed almost twice the risk for developing panic compared to the remainder of the subjects (Odds Ratio (OR) = 1.7, Confidence Interval (CI) = 1.04 - 2.75). In a similar comparison of the top decile of the sample, approximately 20% of the highest decile ASI group experienced a panic attack with a three-times greater risk for experiencing spontaneous panic compared to all others (OR = 2.9, CI = 1.5 - 5.3).

Because a history of spontaneous panic is likely to affect the development of additional panic attacks, we also evaluated the unique contributions of a history of panic and anxiety sensitivity in predicting panic during BCT. History of panic was associated with approximately twice the risk for the development of panic during BCT (χ^2 (1, N = 1020) = 5.20, $p < .05$; OR = 1.9, CI = 1.1 - 3.2). When history of panic and ASI were simultaneously regressed on panic occurrence, both were found to be uniquely predictive of panic (χ^2 (1, N = 1018) = 13.0, $p < .001$, partial r = .11 (ASI), 4.72, $p < .05$, partial r = .07 (History of panic)).

The association between trait anxiety and the development of panic was also assessed. Trait anxiety did not predict panic occurrence (χ^2 (1, N = 1070) = 1.73, $p > .05$, r = .04). When trait anxiety and the ASI were simultaneously regressed, only the ASI significantly predicted panic (χ^2 (1, N = 1065) = 7.87, $p < .01$, partial r = .09). Consistent

Table 3

Anxiety Sensitivity Predicting Anxiety and Mood Symptomatology

Predictors (Time 1)	Beta	partial r	t (c^2)
		Spontaneous Panic - Time 2	
ASI		0.10	(.09) (7.59)**
History of Panic	0.27	(.05)	(3.04)
STAI	0.02	(.03)	(0.62)
ASI X STAI	0.01	(.02)	(0.67)
		Anxiety (BAI) - Time 2	
BAI (Time 1)	0.36	(.49)	17.3***
ASI	0.34	(.19)	4.82***
STAI	-0.03	(-.02)	-0.73
ASI X STAI	0.02	(.04)	1.67
		Depression (BDI) - Time 2	
BDI (Time 1)	0.43	(.52)	18.7***
ASI	0.13	(.07)	2.15*
STAI	-0.02	(-.02)	-0.79
ASI X STAI	0.01	(.03)	1.18
		Hopelessness (BHS) - Time 2	
BHS (Time 1)	0.41	(.50)	17.4***
ASI	0.02	(.01)	0.50

Table 3 (continued)

STAI	0.01	(.01)	0.44
ASI X STAI	0.01	(.01)	0.84

Note. ASI = Anxiety Sensitivity Inventory; BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory, BHS = Beck Hopelessness Scale; STAI = Trait Anxiety scale from the State-Trait Anxiety Inventory.

*p < .05, **p < .01, ***p < .001

with recommendations of Lilienfeld et al. (1993) which suggest that trait anxiety and anxiety sensitivity may act synergistically to predict the development of panic, the interaction between the ASI and STAI was also used to predict panic attacks. The ASI X STAI interaction term was entered hierarchically following the main effects for ASI and STAI. This interaction was not significant (χ^2 (1, N = 1055) = 0.67, p > .05, partial r = .02).

Anxiety Sensitivity Predicting Anxiety and Depression Symptomatology

The relationship between anxiety sensitivity and anxiety and depression symptoms (i.e., BAI, BDI) during BCT was also evaluated (see Table 3 and Figure 1). Two multiple regression analyses controlling for each respective Time 1 symptom level (i.e., BAI or BDI) and trait anxiety indicated that higher levels of anxiety sensitivity were predictive of higher levels of anxiety symptoms (t(1020) = 4.82, p < .0001, partial r = .19). Of those cadets scoring in the upper quartile of the ASI, 34.4% scored in the moderate anxiety range (BAI>15) at Time 2 compared to only 14.6% of for the remainder of the sample. In addition,

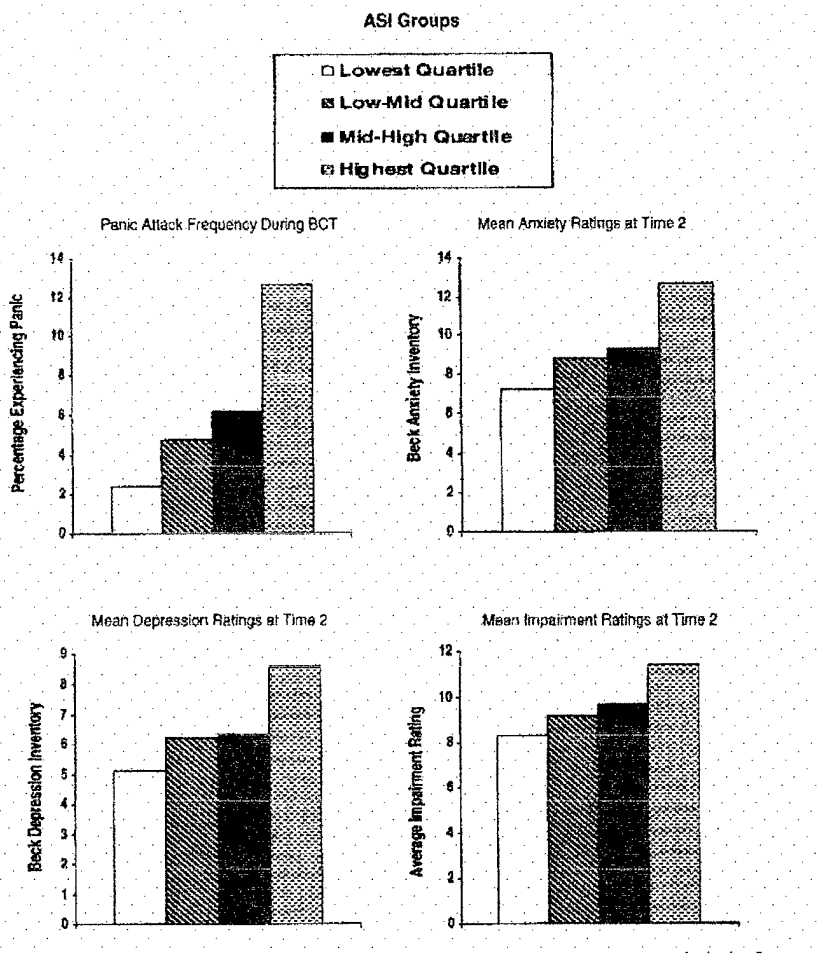

Figure 1. Panic Attacks, Symptomatology, and Impairment across Anxiety Sensitivity Groups based on quartile cutoffs from ASI scores. Means are adjusted for Time 1 scores. Impairment ratings are based on an average score of four questions assessing impairment. There were significant Group effects for all indices (ps $< .001$). Post hoc group comparisons indicated that the Highest quartile ASI group was significantly higher than all other groups on all indices (ps $< .05$).

11.1% of the upper quartile ASI group reported severe anxiety (BAI > 25) compared to only 1.8% of the low ASI group. Higher levels of anxiety sensitivity were also predictive of greater depression symptoms ($t(1017) = 2.15$, $p < .05$, partial $r = .07$), but the ASI was not predictive of hopelessness ($t(1007) = 0.44$, $p > .05$, partial $r = .01$).

Anxiety Sensitivity Predicting Impairment, Disability, Attrition

Apart from its impact on the development of psychopathology, we assessed whether anxiety sensitivity was a risk factor for functional impairment and disability (see Table 4 and Figure 1). Impairment was indexed in terms of self-reported negative influence of anxiety on peer and supervisory relations, physical health, and overall performance. Disability was assessed by number of days at sick call as well as visits to a peer counselor, the counseling center, and the health clinic. Attrition was defined as leaving the academy.

After controlling for the effects of trait anxiety and for Time 1 scores on each respective measure, anxiety sensitivity significantly predicted Time 2 impairment in functioning on all domains including overall performance ($t(1053) = 2.87$, $p < .01$, partial $r = .09$), physical health ($t(1053) = 2.92$, $p < .01$, partial $r = .09$), relations with peers ($t(1053) = 3.12$, $p < .01$, partial $r = .10$) and relations with supervisors ($t(1053) = 2.49$, $p < .05$, partial $r = .08$). In contrast, trait anxiety did not significantly predict impairment in any domain ($ps > .05$). Anxiety sensitivity also significantly predicted three of four disability indices including visits to a peer counselor ($F(1, 1007) = 11.00$, $p < .01$, $r = .13$), visits to the health clinic ($F(1, 1009) = 5.50$, $p < .05$, $r = .11$), and number of days on sick call ($F(1, 1007) = 5.92$, $p < .05$, $r = .11$). Once again, trait anxiety did not significantly predict any disability

Table 4

Anxiety Sensitivity Predicting Impairment, Disability, and Attrition

Predictors (Time 1)	Beta	partial r	$t(c^2)$
		Overall Performance - Time 2	
Overall Performance (Time 1)	0.23	(.17)	5.30***
ASI	0.22	(.09)	2.87**
STAI	0.01	(.01)	0.49
ASI X STAI	0.00	(.01)	0.51
		Peer Relations - Time 2	
Peer Relations (Time 1)	0.20	(.19)	6.18***
ASI	0.06	(.10)	3.12**
STAI	-0.01	(-.03)	-1.15
ASI X STAI	0.00	(.00)	0.08
		Supervisory Relations - Time 2	
Supervisory Relations (Time 1)		0.18	(.23) 7.63***
ASI	0.10	(.08)	2.49*
STAI	0.00	(.00)	0.31
ASI X STAI	0.01	(.01)	0.73
		Physical Health - Time 2	
Physical Health (Time 1)	0.26	(.29)	8.63***
ASI	0.12	(.09)	2.92**
STAI	0.01	(.01)	0.43

Table 4 (continued)

ASI X STAI	0.00	(.00)	0.19
		Peer Counseling - Time 2	
ASI	0.04	(.13)	3.36***
STAI	0.00	(.00)	0.17
ASI X STAI	-0.00	(.00)	0.17
		Counseling Center Visits	
ASI	0.01	(.05)	1.44
STAI	0.00	(.00)	0.14
ASI X STAI	0.00	(.01)	0.63
		Clinic Visits (Physical Illness)	
ASI	0.04	(.11)	2.35*
STAI	0.02	(.02)	1.56
ASI X STAI	0.00	(.00)	0.32
		Sick Call	
ASI	0.04	(.11)	2.43*
STAI	0.02	(.04)	1.64
ASI X STAI	0.00	(.04)	1.57

Table 4 (continued)

		Attrition	
ASI	0.02	(.05)	(1.85)
STAI	0.00	(.00)	(0.32)
ASI X STAI	0.00	(.00)	(0.09)

Note. ASI = Anxiety Sensitivity Index; STAI = Trait Anxiety scale from the State-Trait Anxiety Inventory. *p < .05, **p < .01, ***p < .001.

measure (ps > .05). On average, participants reported less than one visit with a peer counselor (M = 0.4, SD = 1.0) and counseling center (M = 0.0, SD = 0.2), approximately one visit to the health center (M = 0.7, SD = 1.2) and one day of sick call (M = 1.3, SD = 1.4). Those participants scoring in the upper quartile on the ASI reported substantially greater utilization of health care services including approximately twice the number of visits with peer counselors and 50% more time at sick call.

During the course of BCT, 29 participants left the academy. Each cadet completes an out-processing visit at the counseling center prior to leaving. Counseling center records indicated that 38% of cadets left because of a physical condition and only 7% left because of a psychiatric condition, whereas no specific medical diagnosis was provided for the majority of those leaving. Logistic regression using Time 1 ASI and STAI scores to predict attrition were not significant, although a trend (p < .10) was seen for ASI scores only.

Discussion

Early conceptualizations of fear of fear describe it as a consequence of experiencing panic attacks (Goldstein & Chambless, 1978). However, expectancy theory posits that

anxiety sensitivity may precede panic attacks and, therefore, may serve as a premorbid risk factor for the development of anxiety pathology (Reiss, 1991). Empirical evidence for anxiety sensitivity as a risk factor is indirect with the exception being the Maller and Reiss (1992) three year follow-up study. The present study provided an additional prospective test of whether anxiety sensitivity acts as a disposition for anxiety pathology using a substantially larger sample and controlling for other relevant factors including trait anxiety and history of panic. Consistent with hypothesis and the findings of Maller and Reiss, anxiety sensitivity predicted the development of spontaneous panic. The present study also provided an important demonstration that the ASI can uniquely predict the development of clinical phenomena above and beyond the effects of trait anxiety. In conjunction with Maller and Reiss' prospective study, these data provide strong evidence for anxiety sensitivity as a risk factor for the development of panic.

Although psychiatric diagnoses were not assessed in the sample, levels of psychopathology, impairment and disability were evaluated. These findings indicate that higher ASI scores predicted clinically significant symptomatic distress and impairment. For example, 7% of cadets scoring in the upper quartile of the ASI reported moderate to extreme functional impairment from their anxiety and many of these individuals reported multiple panic attacks accompanied by panic-related worry and behavioral impairment. It is likely that some of these individuals, in particular, those reporting severe anxiety and impairment, would meet diagnostic criteria for panic disorder or other anxiety conditions. These findings are also consistent with those of Maller and Reiss (1992) indicating that anxiety sensitivity places individuals at increased risk for anxiety disorders. However, given the reliance on

self-report measures and the absence of diagnostic interviews, caution should necessarily be applied in interpreting symptoms or impairment as diagnoses.

Expectancy theory describes anxiety sensitivity as an amplification factor in fear responding such that individuals with high anxiety sensitivity are more likely to worry about anxiety resulting from a stressor (Reiss & McNally, 1985). It is apparent that anxiety sensitivity should act as a cognitive diathesis that places the individual at risk in the context of stressors. Evaluation of the pathogenesis of panic within a diathesis-stress framework is consistent with the typical history of onset for those developing panic disorder. Often, panic attacks and the formal panic disorder syndrome emerge during highly stressful times (Pollard, Pollard, & Corn, 1989) or when stressors are perceived as relatively uncontrollable (Roy-Byrne, Geraci, & Uhde, 1986). For example, Faravelli and Pallanti (1989) reported that panic disorder patients, compared to nonclinical controls, experienced significantly more negative life events during the year preceding the development of panic, and that the majority of these negative stressors occurred in the month preceding panic.

The present study findings are probably best considered within a diathesis-stress model in which vulnerable individuals were evaluated during exposure to high stress. Despite the limited follow-up interval, it was expected that the five weeks of BCT would be the ideal period to study the development of anxiety pathology as cadets experienced high levels of generally uncontrollable and unpredictable stressors. The relatively high incidence of anxiety pathology during this brief period bear this out. For example, the lifetime prevalence of spontaneous panic prior to BCT was approximately 14% in the sample, but almost half of this number (6%) reported a panic attack during the 5 week period of BCT. Interestingly, cadets generally showed very low levels of anxiety sensitivity. Even the group

experiencing panic attacks reported levels of anxiety sensitivity well below the average for nonclinical samples. This demonstrates that panic attacks can be induced in individuals with relatively few arousal-related fears when substantial stressors are involved.

One of the main limitations of the present study is its reliance on self-report measures for the assessment of panic and anxiety symptoms. Previous work with nonclinical samples suggests that self-report indices may lead to an overreporting of panic attacks (Wilson et al., 1992). Wilson et al. found that relatively few individuals endorsing panic attacks on the PAQ met diagnostic criteria for "clinical" panic. Although validity data for the Panic History Form have shown good agreement with interview assessments of panic, a one year follow-up of a subset of cadets is planned which will include structured diagnostic interviews to provide additional validity data with this sample.

Researchers continue to debate the distinctiveness of anxiety sensitivity and trait anxiety (Lilienfeld, 1996; McNally, 1996). Lilienfeld et al. (1993) have argued that data linking anxiety sensitivity to panic may be accounted for by the shared variance between anxiety sensitivity and trait anxiety. To address this issue, trait anxiety was included as a covariate in relevant statistical analyses. Findings consistently indicated that the ASI accounted for a significant proportion of the variance even when the effects of trait anxiety were partialed out. Conversely, trait anxiety did not predict any major outcome variable. These data are consistent with those of Rapee and Medoro (1994) indicating that anxiety sensitivity accounted for variance beyond the effects of trait anxiety in a series of biological challenge studies. Similarly, we evaluated Lilienfeld et al.'s interpretation of expectancy theory which suggests that the development of panic may be potentiated when high trait anxious individuals also possess high anxiety sensitivity. Findings indicated no significant

anxiety sensitivity and trait anxiety interactions which is consistent with analyses conducted by Taylor (1995) indicating that anxiety sensitivity and trait anxiety did not interact to predict common fears. Our failure to find significant anxiety sensitivity by trait anxiety interactions suggests anxiety sensitivity and trait anxiety do not act synergistically to predict the development of panic.

One notable finding relevant to trait anxiety, however, was its relatively low correlation with the BAI and BDI compared to associations reported in other nonclinical samples (Creamer, Foran, & Bell, 1995). This discrepancy may be due, in part, to BCT-specific factors or to more general factors regarding the military sample. At present, it is unclear what may explain the low level of association. Therefore, conclusions regarding the findings, in particular with respect to trait anxiety, and therefore its relationship to anxiety sensitivity, must be tempered.

The association between anxiety sensitivity and panic attack occurrence, while significant, accounted for only a limited amount of variance. One factor that may underestimate this association is that the sample showed relatively low levels of anxiety sensitivity which is likely to have restricted the variance in the ASI distribution. Scores on the ASI were substantially lower than those reported in other college populations (Telch et al., 1989). Several sample characteristics may account for the low average ASI scores. It would be generally predicted that individuals with high anxiety sensitivity would avoid the military lifestyle (e.g., becoming an Air Force fighter pilot) which, because of its unique demands and risks, is likely to engender anxiety. In addition, the sample was predominantly male, and males have been found to score significantly lower than females on the ASI (Telch et al., 1989). Furthermore, lower ASI scores may be related to a physical health selection

bias for those entering military service. Cadets have undergone screening for physical fitness as well as a comprehensive medical examination. Only cadets in good physical condition who are also free from significant medical conditions are eligible for military service. Interestingly, Schmidt and Telch (in press) found that perceptions of good physical health were associated with lower levels of anxiety sensitivity. Although perceived physical health was not assessed in the present sample, comprehensive physical screening ensures higher than average levels of physical health (Bray, Marsden, & Peterson, 1991; Love & McBride, 1993) which, in turn, is highly associated with better perceived health (Schmidt & Telch, in press).

The observed level of association between anxiety sensitivity and panic suggests that there are other factors involved in the pathogenesis of panic. These factors may independently contribute, or interact with dispositional variables such as anxiety sensitivity, to predict the development of panic. Biological challenge studies have implicated a variety of contextual parameters that affect fearful responding and the development of panic. There is a growing literature to suggest that cognitive factors, such as predictability, perceived control, and perceived safety, influence anxious responding in both clinical (Carter, Hollon, Carson, & Shelton, 1995; Rapee, Mattick, & Murrell, 1986; Sanderson, Rapee, & Barlow, 1989) and nonclinical (Schmidt & Telch, 1994; Telch et al., 1996) populations. Evaluation of other dispositional variables (e.g., attentional focus) and their interaction with contextual factors (e.g., perceived control) may more fully elucidate the relationship between psychological parameters and panic.

Although anxiety sensitivity was originally conceptualized to explain anxiety pathology, several recent studies have found an association between anxiety sensitivity and

depression (Otto, Pollack, Fava, Uccello, & Rosenbaum, 1995; Taylor, Koch, Woody, & McClean, 1996). Findings from the present study extend this work by indicating that anxiety sensitivity may act as a risk factor for the development of depression symptoms. The nature of this linkage, however, is unclear. Negative interpretation and fear of autonomic arousal may directly contribute to the development of depression symptoms, anxiety sensitivity may predict depression because of an association with other dysfunctional cognitive biases linked with depression, or anxiety sensitivity may predict depression because anxiety and depression symptoms covary. Further work is needed to delineate the role of anxiety sensitivity as a vulnerability factor in depression.

The present findings add to the existing knowledge base implicating anxiety sensitivity in the psychopathogenicity of panic and strongly suggest that anxiety sensitivity is a risk factor versus merely an epiphenomenon of developing panic. Because instruction in cognitive behavioral skills can significantly reduce anxiety sensitivity (Telch et al., 1993), this offers an exciting possibility for the implementation of a primary prevention intervention that can effectively anticipate and prevent anxiety and panic reactions among high risk individuals.

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Prospective Evaluation of Psychological Risk Factors as Predictors of Functional Impairment during Acute Stress

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Abstract

Increasing evidence suggests that psychological risk variables (e.g., anxiety sensitivity) may act as premorbid risk factors for the development of physical illness and psychopathology such as clinical anxiety and cardiovascular disease. The principal aim of the present study was to prospectively evaluate the degree to which these factors affected mental and physical health-related impairment and disability. A large nonclinical sample of young adults ($\underline{N}$ = 1296) was prospectively followed during a highly stressful period of time (i.e., military basic training). Consistent with expectation, each of the hypothesized risk factors contributed to the prediction of at least one index of impairment (e.g., visits to health clinic) after controlling for demographic factors, fitness, and history of psychiatric treatment. Moreover, gender interacted with risk factors in predicting outcomes suggesting that females with high anxiety sensitivity, relative to males with high anxiety sensitivity, are particularly prone to develop impairment during stress.

Keywords: Risk Factors, Anxiety, Anxiety Sensitivity, Prospective, Gender

Prospective Evaluation of Psychological Risk Factors as Predictors of Functional Impairment during Acute Stress

There is accumulating evidence that certain specific cognitive and behavioral characteristics will act as risk factors for psychopathology. For example, these psychological risk factors have been found to be elevated in anxiety disorders (1,2). Reductions in these risk factors correspond to decreases in anxiety symptoms (3) as well as decreases in fearful responding to biological challenges (i.e., exposure to substances producing physiological changes) in these patients (4). These risk factors have also been found to predict anxious responding to biological challenge. Challenge studies using nonclinical subjects with no history of anxiety problems have demonstrated that these factors are predictive of fearful responding to hyperventilation, caffeine, and 35% carbon dioxide inhalation (5-9). Longitudinal data also indicate that these risk variables predict the development of anxiety pathology over time (10).

There are three particular psychological risk factors that are promising in terms of playing a significant role in the psychopathogenecity of anxiety: anxiety sensitivity, body vigilance, and discomfort intolerance. Anxiety sensitivity refers to the extent to which an individual believes that symptoms of anxiety or arousal can have harmful consequences (11) and is distinguished from many other cognitive conceptualizations of anxiety because anxiety sensitivity is believed to be a stable trait-like characteristic that may precede the development of pathological anxiety. Individual differences in anxiety sensitivity are hypothesized to emerge from a variety of experiences that ultimately lead to the acquisition of beliefs about the potentially aversive consequences of arousal. Such experiences may include hearing others express fear of such sensations, receiving

misinformation about the harmfulness of certain sensations, witnessing a catastrophic physical event such as a heart attack, and so forth. Thus, anxiety sensitivity constitutes a disposition to developing anxiety and does not require the experience of anxiety or panic in its own development (12).

Body vigilance refers to conscious attention focused on internal bodily sensations and perturbations. The process of monitoring internal states has broad relevance for many theories of emotion and has been described by terms such as visceral perception, autonomic perception, symptom perception, and interoception (13-15). Internal awareness is responsible for important health-relevant behaviors that are both essential and adaptive, such as eating, drinking and resting (13). Thus, internal awareness comprises part of the individual's adaptive monitoring of physiological functioning.

There are individual differences in monitoring and evaluation of internal sensations. Some individuals fail to seek medical care despite symptoms or report no awareness of significant physiological events such as myocardial infarction (16). Others closely monitor internal sensations and repeatedly present for medical evaluation when there is no evidence to suggest organic etiology. In the case of some anxiety disorders, individuals appear to excessively monitor internal sensations because they report very high levels of symptoms and repeatedly seek out medical evaluations despite reassurances from health care providers (17).

Discomfort intolerance is an individual difference variable gauging the degree to which a person tolerates unpleasant physical sensations and has been found to be elevated in patients with anxiety disorders (18). Discomfort intolerance, similar to conceptualizations of pain tolerance/intolerance, is believed to index a psychological

rather than a physiological component underlying the capacity to tolerate and endure pain or discomfort. In contrast to pain tolerance, discomfort intolerance is conceptualized as a broader construct that is related not only to pain but all other types of unpleasant physical symptoms (e.g., pressure, numbness).

Despite growing knowledge of the relationship among these risk factors and anxiety pathology, little is known of their relation to functional impairment particularly in terms of physical health outcomes. It is likely that the same processes that create increased risk for anxiety pathology may also lead to increased risk for other adverse outcomes. For example, recently Asmundson and colleagues have associated one of these variables, anxiety sensitivity, with chronic pain (19).

In this particular study, we were interested in the relationship between psychological risk factors and the development of impairment and disability regardless of mediating factors such as the development of distress symptoms (i.e., anxiety and depression) which will also contribute to impairment. Three corresponding models may be advanced to describe a relationship between these risk factors and impairment. Anxiety sensitivity is believed to impact impairment through its contribution to the development of anxiety and, to a lesser extent, mood symptoms. Higher levels of symptoms, in turn, contribute to impairment and disability. Body vigilance is also believed to play a role in the genesis of anxiety symptoms but is likely to more broadly function in most somaticizing disorders in which an individual is overly attuned to internal bodily symptoms. Discomfort intolerance is believed to produce distress in the context of any stressors that increase unpleasant sensations.

Present Study

The United States Air Force Academy provided an opportunity to evaluate incoming first year students during their initial five weeks of training at the academy. This period, called Basic Cadet Training (BCT), consists of highly regimented training in the context of fairly extreme psychosocial (e.g., isolation from friends and family, constant monitoring and evaluation of behavior) as well as physical (e.g., intense exercise, limited sleep) stressors. These BCT specific factors, in conjunction with the general stressors associated with entering college, make BCT an ideal environment for engendering high levels of stress.

The present study evaluated over one thousand cadets during the beginning and end of BCT to determine whether these risk factors predicted the development of impairment. It was hypothesized that each risk variable would act as a diathesis that would place individuals at risk for the development of impairment in the context of the substantial stressors associated with BCT. More specifically, it was hypothesized that psychological risk factors, independent of demographic factors, fitness, and a history of psychiatric treatment, would predict the development of impairment. Although gender was expected to be generally related to impairment during BCT based on a previous study (20), it was also hypothesized that gender would interact with psychological risk factors such that females, relative to males, scoring high on risk factors, would be at relatively greater risk for impairment.

Method

Participants and Procedure

Participants and procedures are similar to our initial study (10). Participants included 1296 first year undergraduate students (i.e., cadets) from the United States Air

Force Academy (USAFA). Some analyses are based on a lower sample due to missing data.

The BCT period consists of five weeks of training. In general, BCT is designed to continuously expose cadets to a variety of unpredictable and uncontrollable physical and mental stressors. Data for the present study were gathered during a group administration of measures to the entire class of cadets during the first few days of BCT (Time 1) and five weeks after Time 1 at the end of BCT (Time 2). Participants were told that the study was evaluating the impact of BCT on physical and emotional functioning. Prior to the administration of measures, cadets were assured that USAFA officials would not have access to individual identities. Furthermore, code numbers were utilized on all forms to ensure anonymity. Written informed consent was also obtained. The Time 1 assessment battery consisted of measures of anxiety sensitivity, discomfort intolerance, body vigilance, history of mental health treatment, perceived physical fitness, and ratings of impairment. The Time 2 battery included all Time 1 measures plus four additional indices of disability assessing their incidence and frequency during BCT.

Measures

Anxiety Sensitivity Index (ASI). The ASI is a 16-item questionnaire that measures fear of arousal symptoms (21). Each item assesses concern about the possible negative consequences of anxiety symptoms. The ASI has demonstrated adequate internal consistency (22) and test-retest reliability (23). Moreover, the ASI appears to tap fear of anxiety symptoms as opposed to state or trait anxiety (24).

Panic History Form. The Panic History Form is a 4-item screening instrument for assessing history of spontaneous panic attacks (i.e., Have you ever experienced a sudden

and intense surge of fear or anxiety (e.g., a panic attack) in a situation for no apparent reason?), panic attack frequency (i.e., Have you ever experienced four or more panic attacks in the period of one month?), panic-related worry (i.e., Have you ever had a panic attack and subsequently worried about having another for one month or more?), and a general history of psychological and psychiatric treatment (i.e., Have you ever seen a psychologist, psychiatrist, or other mental health professional for treatment?). Expected (i.e., situationally bound or disposed) panic attacks were not assessed. This instrument reliably detects a history of spontaneous panic in nonclinical samples (7,9). Data from a sample of patients presenting for evaluation and treatment indicate that substantial agreement between endorsement of panic attacks and structured interview diagnosis of panic attacks ($\underline{\kappa}$ = .64). Data from nonclinical samples also indicates substantial agreement between self-reported and interview diagnosis of panic attacks ($\underline{\kappa}$ = .58). Compared to structured diagnostic interview, Panic History Form rates of false positive diagnoses of spontaneous panic among nonclinical samples are 45% for participants endorsing at least one spontaneous panic attack, 30% for participants endorsing four panic attacks in a one month period, and 30% for participants endorsing one month or more of panic-related worry. The time frame of this instrument was modified for the Time 2 assessment to cover only the five week BCT period. Participants also rated the number of spontaneous panic attacks experienced during BCT.

Beck Anxiety Inventory (BAI). The BAI is a 21-item measure of anxiety symptoms (25). Each item assesses the degree to which physical or cognitive symptoms of anxiety have affected the individual during the past week. The BAI has been shown to

be a reliable and valid measure of anxiety in a variety of studies using both clinical (coefficient alpha = .92) and nonclinical (coefficient alpha = .91) samples (25,26).

Beck Depression Inventory (BDI). Level of depressive symptoms experienced during the past week was assessed by the 21-item BDI. The BDI is a reliable and well-validated measure of depressive symptomatology (27). Beck, Steer et al. (27) reported high internal consistency (mean coefficient alpha = .81) for nonclinical populations.

Body Vigilance Questionnaire (BVS). The BVS is an four item self-report inventory designed to assess the degree to which an individual attends to internal bodily sensations. Item 1 assesses whether the person typically pays attention to bodily sensations (i.e., I am the kind of person who pays close attention to internal bodily sensations; rated 0 [Not at all like me] - 10 [Extremely like me]). Item 2 assesses perceived sensitivity to changes in bodily sensations (i.e., I am very sensitive to changes in my internal bodily sensations; rated 0 [Not at all like me] -10 [Extremely like me]). Item 3 assesses the average amount of time spent attending to bodily sensations (e.g., On average, how much time do you spend each day "scanning" your body for sensations; rated 0 [No time] -10 [All of the time]). Item 4 involves separate ratings for the amount of attention paid to each of 15 sensations (e.g., heart palpitations, numbness, dizziness, nausea). These sensations include all of the DSM-IV physical symptoms described for panic attacks. Ratings for each of the 15 sensations are averaged to yield one overall score for item 4. The BVS total score is the sum of items 1-4. The BVS possesses good psychometric properties (1).

Discomfort Intolerance Scale (DIS). The DIS is a 6-item measure designed to assess the ability to tolerate discomfort (18). Subjects rate each question (e.g., I am more

sensitive to physical discomfort compared to most people) on a scale ranging from 0 - Not at all like me, to 6 - Extremely like me. Factor analysis of the DIS indicated a two-factor solution with the first factor measuring the ability to tolerate discomfort and pain (e.g., I have a high pain threshold (reverse scored) and the second factor measuring avoidance of discomfort (e.g., I take extreme measures to avoid feeling physically uncomfortable)). Subscales possess adequate internal consistency (Factor 1: $\underline{\alpha}$ = .91; Factor 2: $\underline{\alpha}$ = .72) and adequate stability over three months (Factor 1: $\underline{r}$ = .63; Factor 2: $\underline{r}$ = .66). The overall scale also shows adequate internal consistency ($\underline{\alpha}$ = .75) and was used for all analyses in the present study.

Impairment and Disability. The major outcomes of interest in the present report include self-reported ratings of functional impairment and disability. In order to assess the impact of BCT-related stressors, two self-report questionnaires were constructed. Impairment was assessed using four Likert-format questions (range: 0 = Not at All to 10 = Extremely) rating the degree to which stressors negatively affected: (a) peer relations, (b) supervisory relations, (c) physical health, and (d) overall performance. These questions were administered at both Time 1 and Time 3. Disability was evaluated using four additional questions that assessed the frequency of: (a) peer counseling, (b) counseling center visits, (c) clinic visits for physical illness, and (d) sick call (i.e., absent from activity due to illness). These disability ratings were only assessed at Time 2.

Ratings of physical fitness were assessed using a four item questionnaire (28). A total fitness score is calculated by summing ratings of strength, endurance, flexibility, and speed. Ratings on this questionnaire are associated with measures of aerobic capacity (28).

Results

Relationship among Variables at Time 1

Zero-order correlations among risk factors and impairment variables at Time 1 are presented in Table 1. These correlations suggest only moderate levels of association (r range: .24 - .36) among the three main psychological risk factor variables suggesting that multicolinearity should not be an issue in the later regression analyses.

Table 1

Intercorrelations, Means and Standard Deviations among Major Indices at the Beginning of Basic Training

	1	2	3	4	5	6	7
1. ASI	-						
2. BVS	.36	-					
3. DIS	.33	.24	-				
4. Peer	.20	.09	.08	-			
5. Supervise	.26	.12	.10	.32	-		
6. Physical	.39	.26	.26	.44	.42	-	
7. Overall	.36	.21	.18	.38	.63	.65	-
M	19.8	17.1	15.6	2.2	5.2	3.6	4.5
SD	8.0	7.6	5.7	2.2	2.9	2.7	2.6
α coefficient	.84	.83	.75	-	-	-	-

Note. ASI = Anxiety Sensitivity Index; BVS = Body Vigilance Scale; DIS = Discomfort Intolerance Scale; Impairment Scales assess impairment in Peer and Supervisory Relations, Physical Health, and Overall Impairment.

all ps < .0001, n range = 1224-1289.

Each of these risk factors is significantly, albeit moderately, associated with the impairment ratings. Correlations for the demographic variables indicate that sex and race showed little association with Time 1 impairment ratings. Race was not significantly associated with any index and sex was only associated with physical health (r = .14) and overall impairment ratings (r = .11). Demographics were also not significantly associated with the BVS and the DIS but gender was associated with ASI scores (r = .12). History of mental health care was not significantly associated with any other variable. Higher levels of perceived fitness were negatively associated with impairment in physical health (r = -.14), the ASI (r = -.13) and the DIS (r = -.32).

Psychological Risk Factors predicting Disability during BCT

Stepwise multivariate regression analyses were used to determine the unique contributions of the hypothesized psychological risk variables in predicting disability during the course of BCT. Results of these analyses are presented in Table 2. The analytic procedure involved entering sex and race as an initial step to control for these demographic variables. Age was not used as a predictor because it was deemed that the highly restricted range in this particular sample would not produce meaningful results. Step 2 involved entering perceived physical fitness and any history of mental health treatment. The three psychological risk variables were simultaneously entered for Step 3 and the interactions between the risk factors and sex were entered as the final step.

In terms of measures assessing mental health disability, there were significant effects for sex, the ASI, and also a sex x ASI interaction in predicting visits to a peer counselor. To determine the form of the interaction, rates of peer counseling visits were calculated

Table 2

Predictors of Mental and Physical Disability Outcomes

Entry/Predictors	ΔR^2 Step	t Each Predictor	β	df
		Outcome: Peer Counseling		
Step 1.	.04			6, 921
Sex			5.76****	.19
Race			0.52	.02
Step 2.	.00			8, 915
Physical Fitness			1.80	.06
Hx of Treatment			-1.68	-.09
Step 3.	.01			11, 851
ASI			2.04*	.08
DIS			1.62	.06
BVS			0.36	.01
Step 4.	.02			14, 848
ASI x Sex			3.58***	.37
DIS x Sex			0.38	.04
BVS x Sex			0.96	.09
		Outcome: Hospital Visits		
Step 1.	.02			6, 918
Sex			3.18**	.10
Race			0.87	.04
Step 2.	.00			8, 912
Physical Fitness			-0.99	-.03
Hx of Treatment			-0.69	-.02

Table 2 (cont.)

Step 3.	.01		11, 849
ASI		-0.15	-.00
DIS		1.29	.05
BVS		2.26*	.08
Step 4.	.01		14, 846
ASI x Sex		-0.41	-.04
DIS x Sex		1.13	.12
BVS x Sex		1.86	.19

Outcome: Counseling Center Visits

Step 1.	.02		6, 919
Sex		3.83***	.13
Race		0.40	.01
Step 2.	.00		8, 913
Physical Fitness		-1.06	.03
Hx of Treatment		1.41	.05
Step 3.	.00		11, 850
ASI		0.72	.03
DIS		0.28	.01
BVS		0.01	.00
Step 4.	.01		14, 847
ASI x Sex		2.21*	.24
DIS x Sex		0.22	.02
BVS x Sex		0.18	.02

Table 2 (cont.)

		Outcome: Sick Call	
Step 1.	.04		6, 925
Sex		5.11****	.17
Race		1.22	.04
Step 2.	.00		8, 919
Physical Fitness		-1.60	.05
Hx of Treatment		1.14	.04
Step 3.	.04		11, 855
ASI		-0.03	-.00
DIS		4.57****	.17
BVS		2.17*	.08
Step 4.	.00		14, 852
ASI x Sex		-1.16	-.12
DIS x Sex		-1.37	-.14
BVS x Sex		1.37	.14

Note. ASI = Anxiety Sensitivity Inventory; DIS = Discomfort Intolerance Scale; BVS = Body Vigilance Scale; Hx of Treatmetn = Any history of mental health treatment; β = Standardized β weights are reported for ease of interpretation.

*p < .05, **p < .01, ***p < .001, ****p < .0001.

for participants based on a median split of the ASI across gender. The interaction (see Figure 1) indicated that visits were comparable among low anxiety sensitive individuals whereas high anxiety sensitivity females were more likely to visit a counselor relative to high anxiety sensitivity males. The prediction of visits to the counseling center was

similar in that greater numbers of females reported such a visit and there was a significant sex x ASI interaction. Evaluation of the form of the interaction (using the method described above) indicated a similar effect with high anxiety sensitivity potentiating center visits only for females.

In terms of the prediction of physical health disability, the prediction of hospital visits yielded significant effects for sex, with more females being hospitalized, and an effect for the BVS . Individuals showing higher levels of vigilance to internal arousal cues were more likely to be hospitalized. The prediction of days on sick call was similar with higher numbers of females indicating more days sick, and individuals high in body vigilance indicating more days sick. In addition, the DIS proved to be a fairly strong predictor of sick call with those showing greater intolerance of discomfort taking more sick days.

Psychological Risk Factors predicting Impairment Ratings during BCT

A similar analytic strategy was used to evaluate the relationship among the psychological risk factors and changes in impairment during BCT. The difference in analytic strategy involved entering the Time 1 impairment rating as the first step in each corresponding regression equation. This entry controlled for the initial levels of impairment thereby producing a residualized change variable. These analyses are summarized in Table 3.

Aside from the significant effect of the Time 1 covariate, females reported higher levels of impairment with peer relationships. There were also significant main effects for the ASI and DIS suggesting that these risk factors also produced greater levels of impairment in peer relations. The pattern of effects was essentially identical in the

Table 3

Predictors of Changes in Impairment Outcomes during BCT

Entry/Predictors	ΔR^2 Step	t Each Predictor	β	df
		Outcome: Peer Relations		
Step 1.	.05			1,1137
Time 1 Covariate			7.61****	.22
Step 2.	.01			7,1092
Sex			2.46*	.07
Race			1.29	.04
Step 3.	.00			9, 1088
Physical Fitness			-1.40	-.04
Hx of Treatment			-0.02	-.00
Step 4.	.03			12, 1014
ASI			3.36***	.12
DIS			2.21*	.08
BVS			1.46	.05
Step 5.	.00			15, 1011
ASI x Sex			0.02	.00
DIS x Sex			0.71	.07
BVS x Sex			-0.73	-.07
		Outcome: Supervisory Relations		
Step 1.	.05			1,1136
Time 1 Covariate			8.13****	.23
Step 2.	.01			7,1091
Sex			2.04*	.06

Race		1.11	.04
Step 3.	.00		9, 1087
Physical Fitness		0.16	.00
Hx of Treatment		1.33	.04
Step 4.	.04		12, 1013
ASI		4.11****	.14
DIS		2.11*	.07
BVS		0.57	.02
Step 5.	.00		15, 1010
ASI x Sex		1.50	.14
DIS x Sex		-0.30	-.00
BVS x Sex		-0.73	-.03

Outcome: Physical Health

Step 1.	.10		1,1135
Time 1 Covariate		11.01****	.31
Step 2.	.00		7,1090
Sex		1.53	.04
Race		0.23	.01
Step 3.	.00		9, 1086
Physical Fitness		-0.55	-.02
Hx of Treatment		-0.13	-.00
Step 4.	.03		12, 1013
ASI		3.69***	.13
DIS		2.68**	.09
BVS		1.83	.06
Step 5.	.00		15, 1010
ASI x Sex		1.03	.09

DIS x Sex		0.65	.06
BVS x Sex		0.24	.02
	Outcome: Overall		
Step 1.	.10		1,1132
Time 1 Covariate		11.07****	.31
Step 2.	.01		7,1088
Sex		1.98*	.06
Race		0.93	.03
Step 3.	.00		9, 1084
Physical Fitness		-1.28	-.04
Hx of Treatment		0.74	.02
Step 4.	.03		12, 1011
ASI		3.17**	.11
DIS		2.49*	.08
BVS		0.54	.02
Step 5.	.00		15, 1008
ASI x Sex		-0.38	-.03
DIS x Sex		0.78	.07
BVS x Sex		1.28	.11

Note. ASI = Anxiety Sensitivity Inventory; DIS = Discomfort Intolerance Scale; BVS = Body Vigilance Scale; Hx of Treatmetn = Any history of mental health treatment; β = Standardized β weights are reported for ease of interpretation.

*p < .05, **p < .01, ***p < .001, ****p < .0001.

prediction of supervisory relationships as well as estimation of overall impairment with significant effects for sex, the ASI, and the DIS. The prediction of physical health impairment did not yield an effect for sex but higher levels of ASI and DIS produced higher levels of physical health impairment.

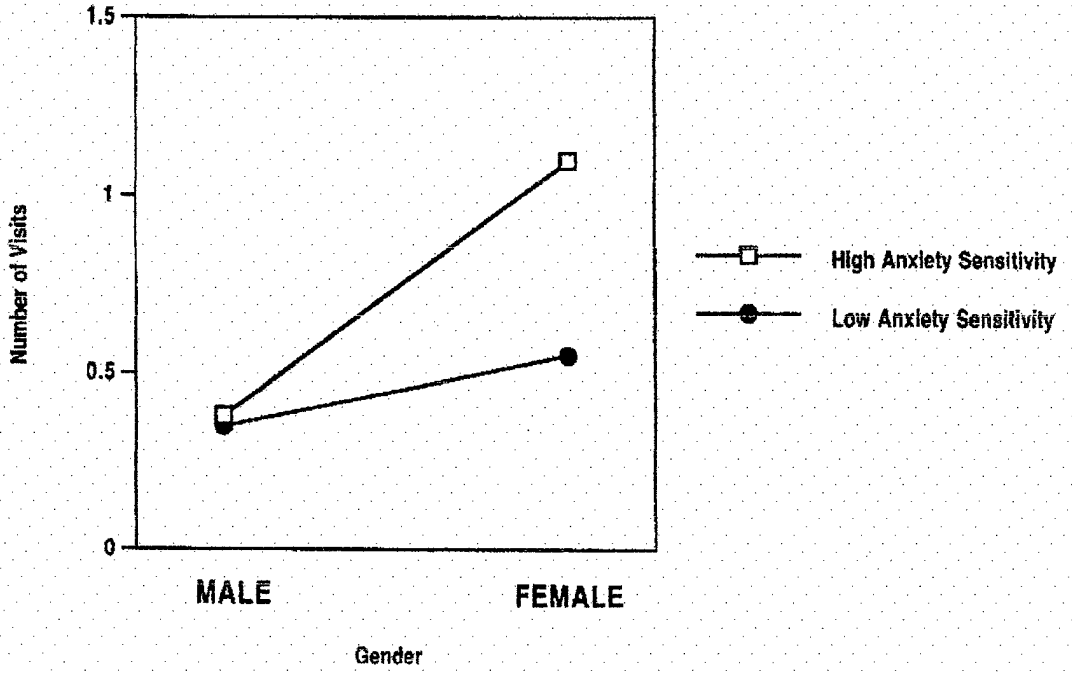

Figure 1. Rate of Peer Counseling Visits across Anxiety Sensitivity (High, Low) and Sex (Male, Female). High Anxiety Sensitivity among Females shows relatively higher rates of visits compared to High Anxiety Sensitivity among Males.

Predictors controlling for Distress Variables

The same analyses were repeated controlling for Time 2 levels of anxiety and mood symptoms as measured by the Beck Anxiety and Depression scales. Surprisingly, the pattern of effects remained relatively stable. The main effect for the ASI dropped out

of the prediction of peer counselor visits but the interaction effect (i.e., sex x ASI) was somewhat stronger ($t(806) = 4.01$, $p < .01$, $\beta = .37$). The predictors of counseling center visits and days sick remained the same. The BVS dropped out of the prediction of hospital visits but an ASI x Sex interaction emerged ($t(803) = 2.02$, $p < .05$, $\beta = .20$) with the interaction form being similar to those previously discussed. Evaluation of the impairment measures produced a more dramatic change in predictors. Sex dropped out of all analyses as did the ASI. However, the DIS continued to be a significant predictor of physical health ($t(950) = 2.12$, $p < .05$, $\beta = .07$) and overall impairment ($t(803) = 2.03$, $p < .05$, $\beta = .06$).

Discussion

The present study is one of the few large prospective efforts aimed at evaluating the role of specific psychological vulnerability factors in the genesis of impairment. Consistent with hypothesis, each of the psychological risk factors predicted the development of impairment after controlling for other relevant variables. Somewhat surprisingly, however, many of these relationships persisted even after controlling for the effects of distress symptoms. This pattern of findings suggests that these psychological risk factors directly impact impairment in addition to impacting impairment vis a vis the development of anxiety and depression symptoms. How might these risk factors directly influence outcome? In general, we would speculate that the presence of these characteristics would lead to an increased likelihood of health-seeking behaviors due to exaggerated concerns about physical and mental well-being. Many individuals with such exaggerated concerns are likely to seek out assistance without any clear awareness of significant anxiety or depression symptoms. Alternatively, these psychological variables

may tap into underlying biological vulnerabilities that make these individuals more prone to need healthcare. Individuals who are naturally less durable or hardy would be more prone to develop negative ideation about their bodies (i.e., anxiety sensivitiy, discomfort intolerance) and would also be more likely to require healthcare.

Gender was evaluated as a moderator variable because of the hypothesis that these vulnerability factors would act differentially in males and females. These hypotheses were partially supported by the emergence of several significant sex x risk factor interactions. It appears that these risk variables potentiate impairment among females particularly in terms of mental health outcomes. Females with high anxiety sensitivity, relative to males, were much more likely to receive peer counseling or visits to the counseling center. This would suggest that females may differentially interpret stress or their stress reactions. Our lab (29) and others have found gender differences in measures of appraisal that indicate potential differences in particular thinking errors (e.g., catastrophizing, overestimation of threat likelihood). For example, Stewart, Taylor and Baker (30) recently found gender differences in anxiety sensitivity in a nonclinical sample of college students. Further investigation is needed to determine the specific nature of these gender differences and the factors that lead to potentiation of impairment in females.

It was expected that the five weeks of BCT would be the ideal period to study the development of stress reactions as cadets experienced high levels of generally uncontrollable and unpredictable stressors. As such, the present study findings are probably best considered within a diathesis-stress model in which vulnerable individuals were evaluated during exposure to high stress. Despite the somewhat unique stressor

experience (military training) and unique population, it is believed that these findings should likely generalize to other acute and chronic stress situations. This assumption has proved to be reasonable in the growing literature focusing on these risk factors and anxiety pathology. However, it remains to be determined whether these risk factors will also be consistently related to impairment indices using other populations.

One obvious study limitation is the exclusive reliance on self-report measures as well as utilization of author-constructed measures of impairment and disability. Because of these limitations, conclusions should be treated with appropriate caution. Moreover, it is worth noting that these findings should not be overinterpreted in light of their relative effect sizes. While the findings were generally consistent with hypotheses, the relative contribution of these psychological risk factors in predicting the outcomes of interest is small in terms of overall variance accounted for. Clearly, there are many other physiological and unassessed psychological factors that also will play a role in the development of impairment and disability in the context of stress. The identified psychological factors represent only one small set of potentially etiologically-significant factors.

It is hoped that the present report will educate the occupational rehabilitation community about some risk factors that they would not have considered in their evaluation of health outcomes. In addition, it is notable that these rating scales are relatively brief, easy to administer and score, and therefore can be readily incorporated into patient assessments. This study offers the preliminary suggestion that these constructs may be useful in elucidating relationships outside the realm of anxiety pathology. Because instruction in skill-based interventions can significantly reduce these

psychological risk factors (3,4), there is an opportunity not only for the implementation of secondary and tertiary interventions but also for the implementation of a primary prevention intervention that can effectively anticipate and prevent stress reactions among high risk individuals.

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Evaluation of Psychological Risk Factors: Prospective Prediction of Psychopathology During Basic Training

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Abstract

Objective: Three theoretically-derived cognitive risk factors were evaluated to determine whether they predicted the development of stress responding in the context of a military training environment (BCT). Method: A large sample of cadets (N = 1401) was prospectively followed over the five week BCT period. Results: All risk factors were found to significantly and independently predict the development of psychopathology and impairment as well as changes in symptoms during basic training. Risk factors conveyed approximately 2-5 times greater likelihood of experiencing clinically significant levels of symptoms at the end of BCT. Conclusions: These data provide strong evidence for three psychological risk factors in the development of anxiety and mood symptoms. Implications for screening and primary prevention are discussed.

Introduction

With reductions in military spending, it has become increasingly important to effectively maintain the quality of military resources and personnel. This emphasis has led to concern in recent years about the effectiveness of current procedures for screening military recruits.[1] No screening procedure however, can prevent some otherwise healthy recruits from developing psychiatric symptoms that can affect both individual and unit performance. New recruits are presented not only with an environment that is novel, but one that is also characterized by a strict social and occupational hierarchy, rigorous physical demands, and an emphasis on conformity and obedience to authority.[2] Previous studies of adaptation to military lifestyle are limited in two respects. First, this work has largely focused on the identification of factors that contribute to retention rates. While the prediction of retention has obvious importance, exclusive focus on adaptation as a dichotomous outcome (i.e., discharge/retention) misses the continuous nature of adaptability. Clinical depression for example, need not lead to discharge but may significantly limit a recruit's productivity. Second, studies in this area have largely focused on general personality traits. Measures such as the Millon Clinical Multiaxial Inventory (MCMI), the Minnesota Multiphasic Personality Inventory (MMPI), and the California Psychological Inventory (CPI),[3,1] while being adequate predictors of retention, assess general maladaptive personality traits that are typically not amenable to treatment. A more useful approach to screening would be to assess predictive and malleable (i.e., treatable) characteristics.

One factor that may compromise military adaptability is the development of psychopathology due to stressors such as Basic Cadet Training (BCT). In particular,

anxiety and depressive symptomatology can manifest in vulnerable individuals under stressful situations[4] such as BCT. Several theories have posited specific risk factors that may serve as vulnerability factors for the development of psychopathology during stress. The current study investigated the predictive value of three of these specific psychological factors, including anxiety sensitivity, reassurance-seeking, and perceived vulnerability.

Anxiety sensitivity refers to the extent to which an individual believes that autonomic arousal can have harmful consequences.[5] For example, individuals with high anxiety sensitivity may believe that shortness of breath signals suffocation or that heart palpitations indicate a heart attack whereas those with low anxiety sensitivity experience these sensations as unpleasant but nonthreatening. Individual differences in anxiety sensitivity are hypothesized to emerge from a variety of experiences that ultimately lead to the acquisition of beliefs about the potentially negative significance of arousal. Such experiences may include hearing others express fear of such sensations, receiving misinformation about the harmfulness of certain sensations, witnessing a catastrophic event such as the fatal heart attack of a loved one, and so forth. Thus, anxiety sensitivity constitutes a disposition to developing anxiety and does not require the experience of anxiety or panic in its own development. Data suggest that high levels of anxiety sensitivity predict the development of panic attacks and other anxiety symptoms.[6]

Coyne's interpersonal theory of depression suggests that interpersonal behaviors, in particular reassurance seeking, play an important role in the development of depression.[7] According to this theory, certain individuals tend to seek reassurance from others as to whether others care about them. Although reassurance is often provided, its

genuineness is questioned and further reassurance is sought. Constant reassurance-seeking leads to aggravation and eventually, to rejection by others, social isolation, and subsequent depression. Reassurance-seeking has been shown to predict depression in at least one prospective study.[8]

Beck's theory of anxiety pathology emphasizes the role of perceived vulnerability in anxiety disorders. Perceived vulnerability refers to a set of beliefs about perceived threat in the world and vulnerability to that threat. More specifically, vulnerability is defined as the perception of omnipresent internal and external dangers over which control is lacking or ineffective.[9] A heightened sense of vulnerability, particularly in the context of high levels of stressors, is hypothesized to result in the development of anxiety and depression and perceived vulnerability has been found to be associated with increased levels of anxiety and depression.[10]

The present study prospectively evaluated whether anxiety sensitivity, reassurance seeking, and perceived vulnerability are associated with the development of anxiety and depression symptoms in a large sample of military recruits. One of the unique aspects of the present study is that the participants were followed over a relatively brief (i.e., five weeks) but highly stressful period of time. The United States Air Force Academy provided an opportunity to evaluate incoming first year students during their initial five weeks of training at the Academy. This period, Basic Cadet Training (BCT), consists of highly regimented training in the context of fairly extreme psychosocial (e.g., isolation from friends and family, constant monitoring and evaluation of behavior) as well as physical (e.g., intense exercise, limited sleep) stressors. These BCT specific factors, in conjunction with the general stressors associated with entering college (e.g.,

first period away from home for many of these young adults), make BCT an ideal environment for engendering psychopathology.

The present study evaluated over one thousand cadets during the first and last weeks of BCT to determine whether these risk factors predicted the development of anxiety pathology. It was hypothesized that anxiety sensitivity, reassurance-seeking, and perceived vulnerability would act as cognitive diatheses that would place individuals at risk for the development of psychopathology in the context of the substantial stressors associated with BCT. More specifically, it was hypothesized that each of these variables would predict the development of symptoms as well as impairment and disability.

Method

Participants and Procedure

Participants included 1401 first year undergraduate students (i.e., cadets) from the United States Air Force Academy (USAFA). From the total pool, 132 cadets were excluded because of incomplete data from the initial (Time 1) assessment. An additional 97 cadets were excluded based on a screening questionnaire that included all items from the MMPI-2 Lie scale.[11] Those scoring greater than 5 on the MMPI-2 Lie scale were excluded to control for defensive responding. Excluded cadets did not differ from the remaining sample in terms of demographics and clinical variables (ps > .05). The remaining participants (N = 1172) were largely male (84%) and Caucasian (84%) with a mean age of 18.0 (SD = 0.9).

The educational requirements of USAFA include military training that begins upon arrival with Basic Cadet Training (BCT). The BCT period consists of five weeks of training divided into two phases. The first phase is characterized by administrative tasks

including placement tests for the academic classes that begin after BCT as well as military indoctrination (e.g., marching, study of military history). The second phase of BCT focuses on physical training. The physical surroundings of the second phase are more extreme as cadets move from the dormitory to tents in the training field. In this context, cadets participate in highly demanding physical activities (e.g., long runs, obstacle courses). In general, BCT is designed to continuously expose cadets to a variety of unpredictable and uncontrollable physical and mental stressors. Cadets are not given schedules and have no access to clocks or watches. They cannot predict whether their next activity will be an academic evaluation, a military exercise, or a five mile run. New stressors are continually introduced to ensure that each cadet is overtaxed.

Data for the present study were gathered during a group administration of measures to the entire class of cadets during the first few days of BCT (Time 1) and five weeks later at the end of BCT (Time 2). Participants were told that the study was evaluating the impact of BCT on physical and emotional functioning. Prior to the administration of measures, cadets were assured that USAFA would not have access to information collected. Furthermore, code numbers were utilized on all forms to ensure anonymity. Written informed consent was also obtained. The Time 1 assessment battery consisted of measures of anxiety sensitivity, vulnerability and reassurance-seeking as well as trait anxiety, anxiety symptoms, depression, and indices tapping the functional impairment created by psychopathology. The Time 2 assessment battery was identical to the Time 1 battery but also included indices of physical and mental disability experienced during BCT. Data on voluntary and involuntary separations (i.e., cadets choosing or forced to leave) from USAFA were obtained from the USAFA Counseling Center.

Measures

Anxiety Sensitivity Index (ASI). The ASI is a 16-item questionnaire that measures fear of arousal symptoms.[12] Each item assesses concern about the possible negative consequences of anxiety symptoms (e.g., It is important to me not to appear nervous). The ASI has demonstrated adequate internal consistency(coefficient alpha = .92)[13] and test-retest reliability (r = .85).[14] Moreover, the ASI appears to tap fear of anxiety symptoms as opposed to state or trait anxiety.[15]

Beck Anxiety Inventory (BAI). The BAI is a 21-item measure of anxiety.[16] Each item assesses a common physical or cognitive symptom of anxiety. The BAI has been shown to be a reliable and valid measure of anxiety in a variety of studies using both clinical and nonclinical samples.

Beck Depression Inventory (BDI). Level of depressive symptoms was assessed by the 21-item BDI. The BDI is a reliable and well-validated measure of depressive symptomatology.[17] Beck and colleagues reported high internal consistency (mean coefficient α = .81) for nonclinical populations.[17]

Impairment, Disability and Attrition. In order to assess the impact of anxiety pathology, a self-report questionnaire was constructed to index impairment and disability. Impairment was assessed using four Likert-format questions (range: 0 = Not at All to 10 = Extremely) rating the degree to which symptoms of anxiety negatively affected: (a) peer relations, (b) supervisory relations, (c) physical health and well-being, and (d) overall performance. The Impairment index showed adequate internal consistency in the present sample (coefficient α = .78). Disability was evaluated using four questions that assessed the frequency of: (a) peer counseling, (b) counseling center visits, (c) clinic

visits for physical illness, and (d) sick call (i.e., absent from activity due to illness). Attrition was assessed from data obtained from the USAFA Counseling Center which processes all cadets separating from the Academy.

Depressive Interpersonal Relationships Inventory-Reassurance Seeking Subscale (DIRI-RS). This 4-item scale measures the tendency to excessively seek reassurance from others (e.g. "Do you frequently seek reassurance from the people you feel close to as to whether they really care about you?"). Considerable data supports the reliability (coefficient α = .90) and construct validity of the scale which is highly correlated to the development of depression in clinical and nonclinical samples.[18,19]

State-Trait Anxiety Inventory (STAI). The STAI is composed of two 20-item scales designed to assess state and trait anxiety. Both scales of the STAI have adequate psychometric properties.[20] Only the trait scale, which measures general levels of anxiety, was utilized in the present study as a control variable.

Vulnerability Scale (VS). The VS is a 8-item measure of perceived threat and physical vulnerability (e.g., I feel that the world is a dangerous place). This measure was derived from Beck's theory of anxiety which postulates vulnerability as the key risk factor in the development of anxiety pathology.[9] The VS possesses adequate reliability (coefficient alpha = .89, test-retest r = .82) and validity and is associated with the development of anxiety symptoms.[10]

Results

Clinical Characteristics at Time 1

Correlations among the major clinical indices are provided in Table 1. The sample generally reported somewhat elevated levels of anxiety (BAI: M = 18.0, SD = 10.6) and

depression (BDI: M = 9.7, SD = 7.2) symptomatology relative to other nonclinical samples. Scores on trait anxiety (STAI: M = 42.5, SD = 5.3) were consistent with normative data for nonclinical undergraduate samples. Levels of the vulnerability factors were low in comparison with other nonclinical college samples (ASI: M = 4.0, SD = 2.9, DIRI-RS: M = 9.1, SD = 4.7, VS: M = 10.1, SD = 7.1). The general pattern of correlations indicates that the risk factors are only modestly associated with each other but that each is significantly associated with symptomatology and impairment (ps < .01) whereas trait anxiety shows a substantially lower level of association with the outcome measures.

The overall level of association between the risk factor variables and peak levels of psychopathology (Time 1) was evaluated using simultaneous regression of all risk factor measures on the BAI and BDI. These analyses indicated that the risk factors accounted for 29% of the variance for anxiety symptoms (ASI: standardized β = .18, $p < .0001$; VS: standardized β = .41, $p < .0001$; RS: standardized β = .10, $p < .0001$) and 33% of the variance for depression symptoms (ASI: standardized β = .10, $p < .0001$; VS: standardized β = .48, $p < .0001$; RS: standardized β = .14, $p < .0001$).

Risk Factors Predicting Changes in Anxiety and Depression Symptomatology

Levels of psychopathology generally decrease over the course of BCT as cadets adapt to the stressors. For example, 47% of cadets report clinically significant levels of anxiety at the start of BCT (Time 1) but only 16% report clinically significant levels of anxiety at the end of BCT (Time 2). High anxiety at Time 1 is an expected reaction to the stressor but those cadets with ongoing anxiety represent a failure to adapt.

Table 1

Intercorrelations, Means and Standard Deviations among Major Indices at Time 1

	ASI	VS	DIRI-RS	STAI	BAI	BDI	IMP
1. ASI	-						
2. VS	.28	-					
3. DIRI-RS	.11	.34	-				
4. STAI	.12*	.09*	.07	-			
5. BAI	.30*	.50*	.25*	.09*	-		
6. BDI	.24*	.56*	.32*	.07	.60*	-	
7. IMP	.21*	.44*	.27*	.01	.51*	.53*	-
M	4.0	10.1	9.1	42.5	18.0	9.7	15.5
SD	2.9	7.1	4.7	5.3	10.6	7.2	8.2

Note. 1 - ASI = Anxiety Sensitivity Index; 2 - VS = Vulnerability Scale, 3 = DIRI-RS = Reassurance Seeking Subscale; 4 = STAI = Spielberger Trait Anxiety Scale; 5 - BAI = Beck Anxiety Inventory; 6 - BDI = Beck Depression Inventory; 7 - Impairment = Total Score from Impairment Scale.

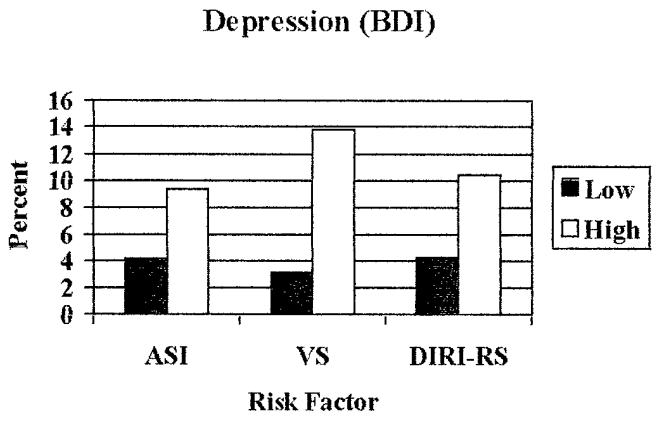
*$p < .01$.

The relationship between each risk factor and changes in anxiety and depression symptoms (i.e., BAI, BDI) during BCT was also evaluated (see Figure 1). These analyses essentially evaluated the level of adaptation (i.e., reduction in symptoms) over time. Multiple regression analyses controlling for each respective Time 1 symptom level (i.e.,

BAI or BDI) and trait anxiety indicated that higher levels of anxiety sensitivity were predictive of higher levels of anxiety symptoms ($t(1020) = 4.82$, $p < .0001$, partial $r = .19$) and greater depression symptoms ($t(1017) = 2.15$, $p < .05$, partial $r = .07$). Logistic regression using a median split of the ASI predicting those subjects falling within the clinically significant range of anxiety (BAI > 15) and depression (BDI > 15) indicate that a high ASI score conveys approximately two times greater risk for developing significant anxiety symptoms (Wald χ^2 (1, N = 1021) = 17.9, Odds Ratio (OR) = 2.0, Confidence Interval (CI): 1.4 - 2.5) and two and a half times greater risk for developing depression symptoms (Wald χ^2 (1, N = 1018) = 14.1, OR = 2.4, CI: 1.5 - 3.7).

Higher scores on the VS were also predictive of higher levels of anxiety symptoms ($t(1020) = 5.64$, $p < .0001$, partial $r = .20$) and greater depression symptoms ($t(1073) = 3.50$, $p < .0001$ partial $r = .11$). Logistic regression analyses using a median split of the VS predicting clinically significant anxiety and depression indicate that a high VS score conveys approximately three times greater risk for developing significant anxiety symptoms (Wald χ^2 (1, N = 1021) = 45.3, OR = 2.8, CI: 2.1 - 3.8) and approximately five times greater risk for developing depression symptoms (Wald χ^2 (1, N = 1074) = 46.1, OR = 4.9, CI: 3.1 - 7.9).

Higher scores on the DIRI-RS were predictive of higher levels of anxiety symptoms ($t(1053) = 3.41$, $p < .0001$, partial $r = .09$) and greater depression symptoms ($t(1070) = 4.57$, $p < .0001$, partial $r = .12$). Similar logistic regression analyses using a



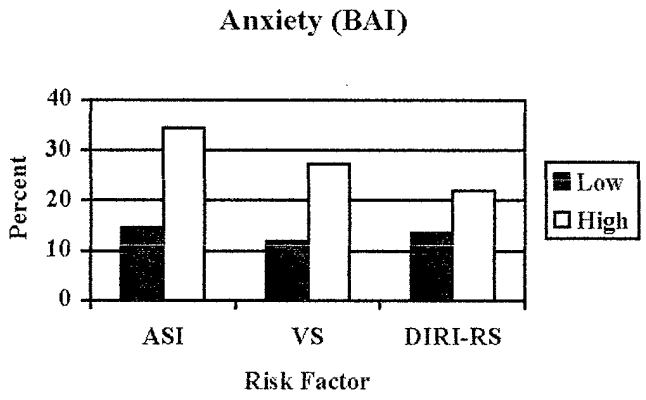


Figure 1. Risk factors (median split) predicting percentage of Cadets exhibiting clinically significant levels of anxiety and mood pathology.

median split of the DIRI-RS predicting clinically significant anxiety and depression indicate that a high DIRI-RS score conveys approximately two times greater risk for developing significant anxiety symptoms (Wald χ^2 (1, N = 1054) = 13.7, OR = 1.8, CI:

1.3 - 2.4) and approximately two and a half times greater risk for developing depression symptoms (Wald χ^2 (1, N = 1070) = 16.9, OR = 2.6, CI: 1.6 - 4.1).

The overall level of association between the predictor variables and changes in levels of psychopathology (Time 2 controlling for Time 1) was evaluated using simultaneous regression of all vulnerability measures on the BAI and BDI. These analyses indicated that the vulnerability measures accounted for 32% of the variance for anxiety symptoms (ASI: standardized β = .09, $p > .001$; VS: standardized β = .14, $p < .0001$; RS: standardized β = .04, $p > .05$; Time 1 BAI: standardized β = .43, $p < .0001$) and 32% of the variance for depression symptoms (ASI: standardized β = .04, $p > .05$; VS: standardized β = .08, $p < .05$; RS: standardized β = .10, $p < .001$; Time 1 BDI: standardized β = .47, $p < .0001$). In both analyses, all risk factors remained as significant predictors suggesting that each uniquely contributes to failure to adapt.

Vulnerability Measures Predicting Impairment, Disability, Attrition

Apart from its impact on the development of psychopathology, we assessed whether each measure served as a risk factor for functional impairment and disability. After controlling for the effects of trait anxiety and for Time 1 scores on each respective outcome measure (covariates), stepwise regression analyses anxiety sensitivity significantly predicted Time 2 impairment in functioning on all domains including overall performance ($t(1053) = 2.87$, $p < .01$, partial r = .09), physical health ($t(1053) = 2.92$, $p < .01$, partial r = .09), relations with peers ($t(1053) = 3.12$, $p < .01$, partial r = .10) and relations with supervisors ($t(1053) = 2.49$, $p < .05$, partial r = .08). Anxiety sensitivity also significantly predicted three of four disability indices including visits to a peer counselor ($F(1, 1007) = 11.00$, $p < .01$, $r = .13$), visits to the health clinic ($F(1, 1009) =$

5.50, $p < .05$, $r = .11$), and number of days on sick call ($F(1, 1007) = 5.92$, $p < .05$, $r = .11$).

The VS significantly predicted Time 2 impairment in functioning on all domains including overall performance ($t(1107) = 6.71$, $p < .0001$, partial $r = .21$), physical health ($t(1105) = 6.46$, $p < .0001$, partial $r = .20$), relations with peers ($t(1107) = 7.85$, $p < .0001$, partial $r = .23$) and relations with supervisors ($t(1106) = 5.73$, $p < .0001$, partial $r = .22$). The VS also significantly predicted two of four disability indices including visits to a peer counselor ($t(920) = 3.69$, $p < .001$, $r = .12$), and number of days on sick call ($t(923) = 2.22$, $p < .05$, $r = .07$).

The DIRI-RS significantly predicted Time 2 impairment in functioning on all domains including overall performance ($t(1114) = 3.16$, $p < .01$, partial $r = .09$), physical health ($t(1118) = 2.40$, $p < .05$, partial $r = .07$), relations with peers ($t(1120) = 3.51$, $p < .001$, partial $r = .10$) and relations with supervisors ($t(1119) = 4.71$, $p < .0001$, partial $r = .14$). The DIRI-RS also significantly predicted two of four disability indices including visits to a peer counselor ($t(932) = 3.87$, $p < .0001$, $r = .13$) and number of days on sick call ($t(935) = 2.87$, $p < .01$, $r = .09$).

During the course of BCT, 29 participants were separated from the academy. Each cadet completes an out-processing visit at the counseling center prior to separation. Counseling center records indicated that 38% of separations were attributed to a physical condition and only 7% were due to a psychiatric condition, whereas the majority of separations indicated no specific medical diagnosis. Logistic regression analyses using Time 1 ASI, VS, and DIRI-RS scores to predict attrition were not significant but there was limited power to detect these effects due to low rates of attrition.

Discussion

The major study hypotheses were supported by the data. Each of the proposed risk factors acted as a significant and independent predictor of distress both at the onset of BCT, when psychopathology was at its highest level, as well as near the termination of BCT when the majority of cadets had essentially adapted to these stressors. Although the level of association between these risk factors and psychopathology was substantial and accounted for approximately 30% of the variance, these data clearly indicate that there are other factors involved in the pathogenesis of anxiety and depression. Moreover, the correlational nature of the study indicates a need for further work using experimental designs. Genetic, physiologic, and other psychological variables will independently contribute, or interact with, these risk factors to predict the development of psychopathology. There are other limitations as well including the fact that measures taken during the initial period of BCT do not represent a true baseline but are taking place during an aroused state.

The present study findings are probably best considered within a diathesis-stress model in which vulnerable individuals possessing a cognitive diathesis were evaluated during exposure to high stress. Despite the limited follow-up interval, it was expected that the five weeks of BCT would be the ideal period to study the development of anxiety and depression as cadets experience high levels of generally uncontrollable and unpredictable stressors. The relatively high incidence of anxiety and mood pathology during this brief period bear this out.

Results of the study reflect positively upon both the adaptability of trainees and the opportunity for training directors to prevent negative psychosocial outcomes during

BCT. Importantly, the vast majority of cadets showed good adaptation to BCT-related stressors as indicated by substantially lower levels of psychopathology at the end of BCT. Although it may be intuitive to someone who has experienced the rigors of BCT, this finding should be welcome in an era when the appropriateness of military training programs is under careful scrutiny by media and lawmakers. Evidently, training of officer candidates in its current state does not exert undue stress upon trainees or cause excessive psychological strain as is evidenced by the fact that only a minority of basic cadets developed significant depression and anxiety symptoms.

The identification of specific risk factors may allow for the future development of brief primary prevention interventions where the risk factors of vulnerable individuals can be modified, thus preventing clinical symptomatology. Such an approach is to be contrasted with a more traditional screening approach, where at-risk individuals may be rejected during the application process or soon after entry into the military. In addition, relatively mild clinical symptomatology as seen in the current study could be addressed without extensive one-on-one treatment. The effectiveness of brief skill-based, group interventions that reduce these risk factors has been demonstrated in a military setting.[21] This previous work suggests that interventions such as those designed for panic disorder and depression could easily be adapted to a nonclinical population. This method of prevention would minimize resource utilization, total cost, and the loss of valuable training time.

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Running Head: ANXIETY SENSITIVITY AND SYMPTOM SPECIFICITY

Anxiety Sensitivity and the Pathogenesis of Anxiety and Depression: Evidence for Symptom Specificity

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Abstract

Expectancy theory posits that anxiety sensitivity (AS) acts as a specific risk factor for the development of anxiety pathology (Reiss, 1991). Previous work suggests that AS is a risk factor for anxiety but several reports have found that AS is also related to depression. The principal aim of the present study was to determine whether anxiety sensitivity acts as a specific vulnerability factor in the pathogenesis of anxiety and depression in both a large nonclinical sample ($\underline{N}$ = 1401) as well as a patient sample ($\underline{N}$ = 53). A covariance analytic strategy indicated that AS possesses symptom specificity with respect to anxiety but is not predictive of depression when accounting for changes in anxiety symptoms. Component analyses suggest, however, that one first-order factor (phrenophobia) is likely to account for the association between AS and depression because it is non-specific (i.e., associated with unique aspects of both anxiety and depression). It is concluded that much of the general association noted between anxiety sensitivity and depression is likely to be due to covariation among symptoms of anxiety and depression.

Anxiety Sensitivity and the Pathogenesis of Anxiety and Depression: Evidence for Symptom Specificity

Anxiety sensitivity refers to the extent to which an individual believes that autonomic arousal can have harmful consequences (Reiss & McNally, 1985). For example, individuals with high anxiety sensitivity may believe that shortness of breath signals suffocation or that heart palpitations indicate a heart attack whereas those with low anxiety sensitivity experience these sensations as unpleasant but nonthreatening. Consistent with cognitive theories of anxiety, the anxiety sensitivity conceptualization posits that cognitive misappraisal is critical for the generation of anxiety. Misinterpretation of bodily sensations leads to a vicious cycle in which faulty interpretation leads to more anxiety as a fearful response to arousal increases the very symptoms that constitute the focus of apprehension. This process may ultimately spiral into full-blown panic as fear feeds upon itself (see McNally, 1990).

Anxiety sensitivity is distinguished from other cognitive conceptualizations because anxiety sensitivity is believed to be a stable trait-like characteristic that may precede the development of pathological anxiety. Individual differences in anxiety sensitivity are hypothesized to emerge from a variety of experiences that ultimately lead to the acquisition of beliefs about the potentially aversive consequences of arousal. Such experiences may include hearing others express fear of such sensations, receiving misinformation about the harmfulness of certain sensations, witnessing a catastrophic event such as the fatal heart attack of a loved one, and so forth. Thus, anxiety sensitivity constitutes a disposition to developing anxiety and does not require the experience of

anxiety or panic in its own development (see Taylor, in press, for a review of anxiety sensitivity).

There is accumulating evidence for anxiety sensitivity as a risk factor for anxiety. Anxiety sensitivity is elevated in panic disorder as well as other anxiety disorders (Taylor, Koch, & McNally, 1992), and anxiety sensitivity decreases with remission of panic disorder symptomatology (Telch et al., 1993). High levels of anxiety sensitivity can precede the development of panic attacks. For example, Donnell and McNally (1990) found that a substantial number of college students with no history of spontaneous panic scored high on the Anxiety Sensitivity Index (ASI) which is the most common measure of anxiety sensitivity. High anxiety sensitivity, independent of a history of panic, has also been found to predict anxious responding to biological challenge (i.e., exposure to substances or procedures that result in physiological changes). Challenge studies using nonclinical subjects with no history of spontaneous panic have demonstrated that anxiety sensitivity is predictive of fearful responding to hyperventilation, caffeine, and 35% carbon dioxide inhalation (Donnell & McNally, 1990; Harrington, Schmidt, & Telch, 1996; Schmidt & Telch, 1994; Rapee & Medoro, 1994; Telch, Silverman, & Schmidt, 1996).

Several prospective studies evaluating nonclinical samples have also suggested anxiety sensitivity acts as a risk factor for the subsequent development of anxiety pathology. Maller and Reiss (1992) conducted a three year follow-up study using a nonclinical sample of college students originally assessed with the ASI. Scores on the ASI were predictive of the frequency and intensity of panic attacks during the follow-up period. Schmidt, Lerew, and Jackson (1997) found that anxiety sensitivity was predictive

of the development of spontaneous panic attacks and anxiety symptoms during military basic training. Taken together, laboratory and prospective studies provide converging evidence for anxiety sensitivity as a risk factor in the development of anxiety pathology.

Recently, several reports have found an association between anxiety sensitivity and depression (Otto, Pollack, Fava, Uccello, & Rosenbaum, 1995; Taylor, Koch, Woody, & McLean, 1996). Otto et al. (1995) reported increased ASI scores in patients with major depression. Evaluation of several cohorts of depressed patients indicated significant elevations when compared to nonclinical controls as well as available norms for nonclinical samples (Peterson & Reiss, 1987). Scores for depressed participants were somewhat lower than patients with panic disorder but were comparable to other anxiety disorders such as generalized anxiety disorder, social phobia, and specific phobia.

Taylor et al. (1996) also found ASI scores to be elevated in patients with major depression. In addition, patients with panic disorder with co-occurring major depression showed higher scores ($M = 40.3$, $SD = 11.3$) compared to those panic disorder patients without depression ($M = 31.4$, $SD = 9.6$). A principal components analysis (PCA) of the ASI yielded a three factor solution that suggested that fear of publicly observable symptoms and fear of bodily sensations were associated with anxiety-related measures but not with depression measures. Conversely, fear of loss of cognitive control (i.e., phrenophobia) was associated with depression but not anxiety measures.

One prospective study of anxiety sensitivity in the context of acute stress also suggested a linkage between anxiety sensitivity and depression symptoms. Schmidt et al. (1997) found that the ASI was a significant predictor of changes in depression symptoms during military basic training. Higher levels of anxiety sensitivity were associated with

higher levels of depression even after controlling for the effects of trait anxiety and a history of spontaneous panic. Thus, there is accumulating evidence to suggest that anxiety sensitivity, or some aspect of anxiety sensitivity such as phrenophobia, may be a risk factor for depression.

Identification of Vulnerability Factors in Psychopathology Research

Garber and Hollon (1991) have provided three criteria for demonstrating, insofar as possible, whether a variable serves as a vulnerability factor in non-experimental psychopathology research (such as the three studies indicating an association between anxiety sensitivity and depression). First, a vulnerability factor and an outcome should covary. Second, a vulnerability factor should temporally precede an outcome. Third, the relationship between the vulnerability factor and an outcome should be non-spurious (e.g., should not be better accounted for by a third variable). Diagnostic and symptom specificity have been described as specific cases of non-spuriousness (Hollon, Kendall, and Lumry, 1986). This refers to the idea that any variable hypothesized to have causal status regarding a psychopathological syndrome should be relatively specific to that syndrome, and should not covary with other phenomenon believed to be distinct from the syndrome of interest.

The specificity criterion for non-spuriousness may be considered overly stringent but it is useful in that it focuses on one of an impressive array of sources of spuriousness (see Joiner, Katz, & Lew, in press). It is also particularly applicable for the construct of anxiety sensitivity which has been proposed to be a specific risk factor for anxiety but not as a general risk factor for other forms of psychopathology. Thus, while the association between anxiety sensitivity and depression is interesting, it poses a conceptual difficulty

in relation to the issue of non-spuriousness. If anxiety sensitivity is a vulnerability factor for both anxiety and depression, it loses its conceptual specificity as a risk factor for anxiety and may be better considered to be a general risk factor for psychopathology. On the other hand, perhaps anxiety sensitivity possesses predictive specificity generally, while some first-order factor of anxiety sensitivity accounts for its association with depression. There have been a variety of factor analytic evaluations of the ASI, and these reports appear to converge on the idea that anxiety sensitivity is unifactorial at a higher level but multifactorial at a lower level (Cox, Parker, & Swinson, 1996). Moreover, the Taylor et al. (1996) report suggests that lower-order phrenophobia factor may largely account for the association between anxiety sensitivity and depression.

The present study sought to extend research on the association between anxiety sensitivity and depression. There are several plausible explanations for this association. Otto et al. (1995) speculate that the same cognitive distortions that are characteristic of depression may be associated with the attributional style described by anxiety sensitivity. For example, cognitive biases leading to catastrophizing or likelihood overestimation may be common to anxiety sensitivity and depression. Taylor et al. (1996) suggest that the association may be largely due to the fears tapped by the ASI's lower-order factor assessing phrenophobia. Phrenophobia may act as a specific risk factor for the development of depression because depression typically includes impairment in concentration and decision making difficulty. Accordingly, some individuals with specific fears regarding these symptoms (i.e., phrenophobics) would be more likely to become distressed in their context. There is also the possibility that, regardless of the underlying mechanism, anxiety sensitivity's link with depression is due to the fact that

symptoms of anxiety and depression covary. In this case, anxiety sensitivity would be expected to predict depression only insofar as it is a risk factor for anxiety which will be accompanied by depression.

In order to further evaluate these hypotheses, we examined the relationship between anxiety sensitivity and depression in the context of Garber and Hollon's (1991) non-spuriousness criterion. Previous work only meets two of the three criteria for determining whether anxiety sensitivity is a risk factor for depression in that these variables have been found to covary (Otto et al., 1995; Schmidt et al., 1997; Taylor et al., 1996) and that anxiety sensitivity has been found to be temporally precedent to depression symptoms (Schmidt et al., 1997). None of these studies have evaluated non-spuriousness in relation to predictive specificity. It was hypothesized, in line with expectancy theory, that anxiety sensitivity would be found to act as a specific risk factor for anxiety pathology (i.e., anxiety sensitivity would possess symptom specificity for anxiety). In line with Taylor et al., we also evaluated components (i.e., first-order factors) of anxiety sensitivity. It was predicted, consistent with the findings of Taylor et al., that only the phrenophobia component of anxiety sensitivity would possess symptom specificity for depression. In order to determine the generalizability of these findings, we evaluated these hypotheses in both nonclinical and clinical samples.

Method

Participants and Procedure

Study 1 - Nonclinical Sample. Participants included 1401 military recruits undergoing a five week basic training period at a service academy (participant and procedural details of the study are presented elsewhere, see Schmidt, Lerew, & Jackson,

1997). In general, basic training (BT) is designed to be a highly stressful experience. Data were gathered during a group administration of measures to the entire cohort during the first few days of BT (Time 1) and five weeks later at the end of BT (Time 2). Participants were told that the study was evaluating the impact of BT on physical and emotional functioning. Prior to the administration of measures, participants were assured that the military would not have access to information collected. Furthermore, code numbers were utilized on all forms to ensure anonymity. Written informed consent was also obtained. Both assessment batteries included of measures of anxiety sensitivity, anxiety symptoms, depression and hopelessness.

Study 2 - Clinical Sample. Participants included patients with panic disorder (N = 53) participating in a cognitive behavioral treatment outcome study (see Schmidt, Staab, Trakowski, & Sammons, 1997 for a detailed description of the treatment procedures). Patients met the following entry criteria: (a) principal DSM-IV Axis I diagnosis of panic disorder with or without agoraphobia, (b) no change in medication type or dose during the eight weeks prior to treatment, (c) no evidence of serious suicide intent, (d) no evidence of current substance abuse, (e) no evidence of current or past schizophrenia, bipolar disorder, or organic mental disorder. Subjects' mean age was 33 with a range from 21 to 56. The majority of subjects were female (67%) and Caucasian (79%). Participants were drawn from a pool of subjects presenting at an outpatient psychiatry clinic at a tertiary care hospital. Diagnostic assessment was based on a face-to-face structured clinical interview using the SCID- I/P (First, Spitzer, Gibbon, & Williams, 1994). Randomly selected videotaped interviews from our laboratory have demonstrated

acceptable kappa coefficients for interrater agreement for all Axis I diagnoses (see Schmidt, Lerew, & Trakowski, 1997; Schmidt, Trakowski, & Staab, in press).

On average, participants reported an eight year history (SD = 10.9) of panic disorder with 27% meeting DSM-IV criteria for at least one other anxiety disorder diagnosis and 22% meeting criteria for a mood disorder diagnosis. Total panic attack frequency was 10.3 (SD = 13.5), and unexpected panic attack frequency was 4.1 (SD = 7.5), for the past month. Forty-nine percent of the subjects were taking medications for their anxiety condition. Breakdown by medication type indicated that 25% were taking only benzodiazepines, 12% were taking only antidepressants, and 12% were taking both benzodiazepines and antidepressants.

Measures of anxiety sensitivity, anxiety, and depression were obtained from pretreatment and postreatment assessments.

Measures

Anxiety Sensitivity Index (ASI). The ASI is a 16-item questionnaire that measures fear of arousal symptoms (Peterson & Reiss, 1987). Each item assesses concern about the possible negative consequences of anxiety symptoms. The ASI has demonstrated adequate internal consistency (Telch, Shermis, and Lucas, 1989) and test-retest reliability (Maller & Reiss, 1992). Moreover, the ASI appears to tap fear of anxiety symptoms as opposed to state or trait anxiety (see McNally, 1994).

Beck Anxiety Inventory (BAI). The BAI is a 21-item measure of anxiety symptoms (Beck, Epstein, Brown & Steer, 1988). Each item assesses the degree to which physical or cognitive symptoms of anxiety have affected the individual during the past week. The BAI has been shown to be a reliable and valid measure of anxiety in a

variety of studies using both clinical (coefficient alpha = .92) and nonclinical (coefficient alpha = .91) samples (Beck et al., 1988; Borden, Peterson, Jackson, 1991).

Beck Depression Inventory (BDI). Level of depressive symptoms experienced during the past week was assessed by the 21-item BDI. The BDI is a reliable and well-validated measure of depressive symptomatology (see Beck, Steer, & Garbin, 1988 for a review).

Beck Hopelessness Scale (BHS). Level of hopelessness was assessed by the 20-item BHS. The BHS is a reliable and validated measure of pessimism about the future (Beck, Weissman, Lester, & Trexler, 1974). Beck, Steer, Sanderson, and Skeie (1991) reported high internal consistency for clinical and nonclinical populations (average coefficient alphas in .80s).

Results

Study 1. Symptom Specificity in a Nonclinical Sample

Means, standard deviations, test-retest correlations, and alpha internal consistency coefficients for all variables are presented in Table 1, which also presents the zero-order correlations among all measures. Test-retest correlations and alpha internal consistency coefficients were acceptable for all measures. Overall, the sample reported levels of symptomatology consistent with normative data for nonclinical undergraduate populations except for elevations of anxiety symptomatology (BAI: M = 18.0, SD = 10.6) which is consistent with the stressful environmental context. Scores on the other self-report measures suggested that depression (BDI: M = 9.7, SD = 7.2), and hopelessness (BHS: M = 2.9, SD = 2.9) were generally within the normal range of functioning. Levels of anxiety sensitivity were low (ASI: M = 4.0, SD = 2.9) in comparison with other

Table 1

Intercorrelations, Means and Standard Deviations among Major Indices at Time 1 (below Diagonal) and Time 2 (above Diagonal)

	1	2	3	4	5	6	7	M	SD
1. ASI	(.65)	.31*	.19*	.08*	.80*	.65*	.68*	3.8	5.4
2. BAI	.30*	(.53)	.53*	.25*	.20*	.22*	.22*	9.6	7.3
3. BDI	.24*	.60*	(.55)	.55*	.14*	.17*	.13*	6.4	6.2
4. BHS	.10*	.32*	.58*	(.48)	.05	.11*	.01	2.2	2.3
5. Factor I	.83*	.22*	.15*	.05	(.61)	.41*	.56*	3.1	2.2
6. Factor II	.63*	.32*	.23*	.17*	.42*	(.50)	.33*	0.3	0.8
7. Factor III	.67*	.27*	.31*	.20*	.54*	.31*	(.60)	0.4	0.9
M		4.0	18.0	9.7	2.9	3.3	0.4	0.4	
SD		2.9	10.6	7.2	2.9	2.1	0.8	0.9	
□ coefficient	.66	.91	.86	.81	.57	.45	.47		

Note. Correlations between scores on each measure at Time 1 and Time 2 appear in the diagonal. 1 - ASI = Anxiety Sensitivity Index; 2 - BAI = Beck Anxiety Inventory; 3 - BDI = Beck Depression Inventory; 4 - BHS = Beck Hopelessness Scale.

*$p < .01$.

nonclinical college samples (see Telch, Lucas, & Nelson, 1989) but is likely to be the result of a selection bias for individuals who enter the military. The general pattern of correlations indicates that anxiety sensitivity is significantly associated with both anxiety and depression symptoms at both Time 1 and Time 2 (ps < .01).

Symptom Specificity of Anxiety Sensitivity.

Consistent with recommendations of Kendall and Ingram (1989), we used a covariance strategy to assess predictive specificity (see Joiner, Katz, & Lew, in press, for a review). Two regression equations were constructed. The assumption of homogeneity of covariance was tested and met (cf. Joiner, 1994).

For the first equation, with T2 BAI scores as the dependent variable, T1 BAI scores were entered into the equation, thereby creating residualized change scores in anxiety from T1 to T2. Next, T1 and T2 BDI scores were inserted into the equation to control for changes in depression. Finally, ASI scores were entered into the equation. This approach allows for the examination of the relation of anxiety sensitivity to anxiety symptoms beyond the effects of depression symptoms.

For the second equation, a similar approach was taken, except that T2 BDI scores served as the dependent variable, T1 BDI scores were entered into the equation first, T1 and T2 BAI scores were entered next, and, finally, ASI scores were entered. This allows for the assessment of the relationship between anxiety sensitivity to depression beyond the effects of anxiety.

Results indicated that anxiety sensitivity was specifically related to anxiety but not depression. The ASI was a significant predictor of BAI change scores beyond BDI change ($pr = .09$, $t\ (1010) = 3.74$, $p < .001$); ASI scores were not related to BDI change scores beyond changes in anxiety ($pr = -.00$, $t\ (1010) = -0.14$, $p > .05$).

A similar set of analyses was conducted examining hopelessness symptoms as measured by the BHS. Once again, results supported the hypothesis that ASI would be a specific predictor of anxiety. The ASI was a significant predictor of BAI change scores

beyond BHS change (pr = .12, t (987) = 4.57, p < .0001); ASI scores were not related to BHS change scores beyond changes in anxiety (pr = -.01, t (987) = 0.44, p > .05).

Symptom Specificity among Anxiety Sensitivity Components (Factors).

The same covariance strategy described above was used to evaluate the relationship among the three ASI first-order factors described by Taylor et al. (1996) and anxiety and depression symptoms. Each of these primary-order factors was evaluated separately.

Fear of publicly observable symptoms (Factor I) was specifically related to anxiety but not depression. Factor I was a significant predictor of BAI change scores beyond BDI change (pr = .07, t (1019) = 3.10, p < .01) and beyond BHS change (pr = .11, t (993) = 3.91, p < .0001). Factor I was not related to BDI scores (pr = .00, t (1019) = 0.18, p > .05) or BHS scores (pr = -.00, t (993) = -0.02, p > .05) after controlling for BAI change.

Similarly, Fear of bodily sensations (Factor III) was specifically related to anxiety but not depression. Factor III was a significant predictor of BAI change scores beyond BDI change (pr = .08, t (1019) = 3.11, p < .01) and beyond BHS change (pr = .10, t (992) = 3.51, p < .001). Factor III was not related to BDI scores (pr = -.03, t (1019) = -1.11, p > .05) or BHS scores (pr = -.00, t (992) = -0.15, p > .05) after controlling for BAI change.

Phrenophobia (Factor II) did not show symptom specificity. Instead, Factor II was a predictor of both anxiety and depression symptoms. Factor II was a significant predictor of BAI change scores beyond BDI change (pr = .07, t (1025) = 2.65, p < .01) and beyond BHS change (pr = .09, t (997) = 3.10, p < .01). Factor II was marginally related to BDI

Table 2

Anxiety Sensitivity Predicting Anxiety and Mood Symptoms in a Nonclinical Sample

Order of Entry

Predictors		Standardized Beta	t	df
		Time 2 - Anxiety Symptoms (BAI Scores)		
1.	T1 BAI	.54	21.25****	1, 107
2.	T1 BDI	-.07	-2.13*	2, 102
	T2 BDI	.42	14.49****	
3.	T1 ASI	.09	3.74***	1, 101
		Time 2 - Depression Symptoms (BDI Scores)		
1.	T1 BDI	.55	21.90****	1, 109
2.	T1 BAI	-.17	-5.14****	2, 102
	T2 BAI	.41	14.52****	
3.	T1 ASI	-.00	-0.14	1, 101
		Time 2 - Hopelessness (BHS Scores)		
1.	T1 BHS	.52	19.86****	1, 106
2.	T1 BAI	-.06	-1.93	2, 998
	T2 BAI	.18	5.63****	
3.	T1 ASI	-.01	-0.45	1, 98

Note. T1 = Time 1; T2 = Time 2; ASI = Anxiety Sensitivity Inventory; BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory, BHS = Beck Hopelessness Scale.

*p < .05, **p < .01, ***p < .001.

Table 3

Intercorrelations, Means and Standard Deviations among Major Indices at Pretreatment (below Diagonal) and Posttreatment (above Diagonal) for Patients with Panic Disorder

	ASI	BAI	BDI	I	II	III	M	SD
1. ASI	(.57)	.46*	.36*	.53*	.48*	.54*	12.9	10.1
2. BAI	.52*	(.50)	.28	.58*	.57*	.47*	8.5	8.5
3. BDI	.46*	.52*	(.67)	.37*	.43*	.28	6.6	6.7
4. Factor I	.95*	.52*	.40*	(.54)	.41*	.49*	9.1	6.9
5. Factor II	.75*	.40*	.24	.59*	(.49)	.25	1.6	1.8
6. Factor III	.89*	.42*	.44*	.83*	.53*	(.56)	2.9	3.2
M		29.6	24.9	15.9	18.8	5.4	8.1	
SD		11.3	14.3	10.4	6.5	3.1	4.4	
□ coefficient	.93	.93	.91	.87	.73	.86		

Note. Correlations between scores on each measure at Time 1 and Time 2 appear in the diagonal. 1 - ASI = Anxiety Sensitivity Index; 2 - BAI = Beck Anxiety Inventory; 3 - BDI = Beck Depression Inventory; 4 - BHS = Beck Hopelessness Scale.

*$p < .01$.

scores (pr = .04, t (1025) = 1.59, p = .11) and was a significant predictor of BHS scores (pr = .08, t (997) = 2.75, p < .01) after controlling for BAI change.

Study 2. Symptom Specificity in a Clinical Sample

Means, standard deviations, test-retest correlations, and alpha internal consistency coefficients for all variables are presented in Table 3, which also presents the zero-order correlations between all measures in the clinical sample. Test-retest correlations and alpha internal consistency coefficients were acceptable for all measures.

The same covariance strategy described above was used to assess predictive specificity of symptoms at posttreatment (see Table 4). Findings paralleled those obtained in the nonclinical sample. Results indicated that anxiety sensitivity was specifically related to anxiety but not depression symptoms. The ASI was a significant predictor of BAI change scores beyond BDI change (pr = .28, t (50) = 2.15, p < .05); ASI scores were not related to BDI change scores beyond changes in anxiety (pr = -.06, t (49) = -0.51, p > .05).

Component analyses of specificity using the three ASI factors also indicated a similar pattern of findings relative to the nonclinical sample although none of these analyses reached statistical significance. Specifically, Factors I and III were the more highly associated with changes in BAI scores beyond BDI change (pr = .19, t (50) = 1.57; pr = .22, t (50) = 1.64; respectively) compared to Factor II (pr = .08, t (50) = 0.69). A reverse pattern was true for the prediction of BDI scores. Factor II was more highly associated with depression changes after controlling for anxiety (pr = .12, t (50) = 1.32) relative to Factors I and III (pr = -.07, t (50) = -0.68; pr = -.08, t (50) = -0.78; respectively).

Table 4

Anxiety Sensitivity Predicting Anxiety and Mood Symptoms in a Clinical Sample

Order of Entry

	Predictors	Standardized Beta	t	df
		Time 2 - Anxiety Symptoms (BAI Scores)		
1.	T1 BAI	.50	4.14***	1, 53
2.	T1 BDI	-.44	-3.04**	2, 51
	T2 BDI	.67	4.86***	
3.	T1 ASI	.28	2.15*	1, 50
		Time 2 - Depression Symptoms (BDI Scores)		
1.	T1 BDI	.66	6.33***	1, 52
2.	T1 BAI	-.24	-2.21*	2, 50
	T2 BAI	.39	4.86***	
3.	T1 ASI	-.06	-0.51	1, 49

Note. T1 = Time 1; T2 = Time 2; ASI = Anxiety Sensitivity Inventory; BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory.

*p < .05, **p < .01, ***p < .0001.

Discussion

Expectancy theory posits that anxiety sensitivity may precede panic attacks and, therefore, may serve as a premorbid risk factor for the development of anxiety pathology

(Reiss, 1991). Consistent with theory, converging evidence from experimental and quasi-experimental studies firmly supports the role of anxiety sensitivity in the pathogenesis of anxiety (see Taylor, in press for a review). Recent evidence also describes an association between anxiety sensitivity and depression but this association has not been adequately evaluated. The present report suggests that anxiety sensitivity possesses symptom specificity with respect to anxiety pathology when a covariance strategy is utilized (i.e., examining changes in anxiety when controlling for changes in depression and vice versa). In general, these findings indicate that much of the association noted between anxiety sensitivity and depression is likely to be due to covariation among symptoms of anxiety and depression.

The present study is the first to comprehensively evaluate anxiety sensitivity in the context of criteria necessary for establishing a risk factor in quasi-experimental research designs (i.e., covariance, temporal antecedence, non-spuriousness). Findings are consistent with previous work indicating that anxiety sensitivity is a risk factor for anxiety. Importantly, the present study adds significantly to previous quasi-experimental studies such as our recent prospective study of anxiety sensitivity (Schmidt et al., 1997) as it suggests that anxiety sensitivity meets the critical third criterion (i.e., non-spuriousness) in its determination as a vulnerability factor. Despite the fact that there are many sources of spuriousness, symptom specificity represents a fairly conservative and, therefore, stringent test of this criterion.

On the other hand, the relationship between anxiety sensitivity and depression failed to meet the non-spuriousness criterion. One question that may be raised is whether the symptom specificity criterion is too stringent because of the well-established overlap

between anxiety and depression. Zero-order correlations among these measures attest to the statistically significant and often substantial level of overlap (r range: .25 - .60). We would argue, however, that most current views of anxiety and depression suggest that these are unique and discriminable phenomena. Prominent current views of psychopathology, such as the tripartite model (Clark & Watson, 1991) indicate that anxiety and depression have both overlapping and unique features. Considerable research evaluating the tripartite model indicates a factor specific to anxiety (i.e., physiological hyperarousal) as well as a unique depression factor (i.e., anhedonia). In this respect, the covariance strategy employed in the present report provided a means for partialling out the common features of anxiety and depression (i.e., negative affect) to allow for a more fined tune analysis of their unique features.

In terms of restating these findings more specifically, anxiety sensitivity does not appear to act as a risk factor for the "pure" or unique aspects of depression but is a risk factor for the unique aspects of anxiety. In regard to the tripartite model, anxiety sensitivity may not be a specific risk factor for symptoms of anhedonia but may be a risk factor for symptoms of hyperarousal. This is in keeping with anxiety sensitivity conceptualizations which predict that the key risk outcomes (i.e., panic attacks and panic disorder) for individuals with high anxiety sensitivity are perhaps quintessential representations of hyperarousal. This does not mean, however, that anxiety sensitivity does not amplify some of the associated symptoms of depression in the context of anxiety. Again, in terms of the tripartite model, anxiety sensitivity may amplify non-specific levels of negative affect but probably does not directly influence anhedonia. Further evaluations of anxiety sensitivity in the context of the tripartite model of anxiety

and depression may further elucidate some of these distinctions (cf. Joiner, Steer, A.T. Beck, Schmidt, & Rudd, 1997).

Can you have your cake and eat it too? Analyses of the anxiety sensitivity first-order factors indicate that this may be the case. In other words, anxiety sensitivity generally retains its status as a specific risk factor for anxiety but one of the first-order factors of anxiety sensitivity appears to be linked with depression. The phrenophobia aspect of anxiety sensitivity was found to be associated with both the unique features of anxiety as well as the unique features of depression. It would seem that these findings should be of particular interest to depression researchers evaluating cognitive risk factors for mood pathology.

In contrast to Taylor et al. (1996) who conclude that phrenophobia is a depression-specific measure that is unrelated to anxiety, findings from the present report, particularly among the patient sample, indicate that fear of loss of cognitive control is related to both depression and anxiety. Phrenophobia is also conceptually consistent with anxiety sensitivity in that cognitive symptoms such as poor concentration, and other presumably related symptoms such as depersonalization and derealization, could lead to specific fears. We believe that it is premature to conclude that this construct is unrelated to anxiety pathology. Although the pattern of correlations presented by Taylor et al. is generally comparable to those in the present report, Taylor et al. report substantially higher levels of association between phrenophobia and depression. One explanation for the discrepancies across studies may be due to problems with the reliability of this relatively short (i.e., three item) scale. We reiterate recommendations of Taylor et al. in regard to the expansion of items for a measure of phrenophobia. For example, the

relatively common arousal symptoms of derealization and depersonalization would appear to be good candidates for an expanded phrenophobia scale. A longer and presumably more reliable measure is likely to be necessary for research specifically evaluating the phrenophobia construct.

The present findings add to the growing knowledge base implicating anxiety sensitivity in the psychopathogenicity of anxiety and strongly suggest that anxiety sensitivity is a specific risk factor for anxiety pathology. The present findings have also clarified the linkage between anxiety sensitivity and depression but further work is needed to delineate the role of phrenophobia as a vulnerability factor in depression.

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Effects of Heart Rate Feedback on Estimated Cardiovascular Fitness in Patients with Panic Disorder

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Abstract

Objective: Psychological parameters believed to affect estimations of cardiovascular fitness were examined in patients with panic disorder and nonclinical controls. Method: Fifty four participants [panic disorder patients (n = 27) and age and sex matched nonclinical controls (n = 27)] completed a cycle ergometer test and were compared on the basis of estimated $VO_{2\,max}$. Participants were randomly assigned to experimental conditions in which they received heart rate feedback or no feedback during the test. Results: Patients with panic disorder exhibited lower $VO_{2\,max}$ and decreased exercise tolerance (i.e., were more likely to discontinue the test) than nonclinical controls. Furthermore, individuals with high anxiety sensitivity (i.e., a fear of autonomic arousal), but not a panic disorder diagnosis per se, achieved significantly lower $VO_{2\,max}$ when provided with heart rate feedback. Moreover, diagnostic status interacted with levels of anxiety sensitivity to predict $VO_{2\,max}$. Conclusions: Patients with panic disorder display poorer cardiovascular fitness after controlling for anxiety and other factors that underestimate performance during fitness testing.

Effects of Heart Rate Feedback on Estimated Cardiovascular Fitness in Patients with Panic Disorder

Patients with panic disorder appear to be at increased risk for cardiovascular disease (1,2,3). Coryell and colleagues found approximately a twofold increase in cardiovascular mortality in patients with panic disorder compared to the general population (2,3). This finding appears to be diagnostically specific, as excess cardiovascular mortality was not found in patients with other anxiety disorders such as obsessive-compulsive disorder (4). Weissman et al.'s (1) assessment of cardiovascular problems from the New Haven portion of the ECA survey found that a lifetime diagnosis of panic disorder was associated with higher risks for hypertension, myocardial infarction, and stroke relative to those with no history of psychiatric disorders. Moreover, those with panic disorder showed increased risk for stroke relative to patients with other psychiatric conditions.

The linkage between coronary heart disease (CHD) and anxiety has also been demonstrated for specific symptoms of anxiety. Kawachi and colleagues (5,6,7) reported an increased risk of fatal CHD with higher levels of self-reported worry and phobic anxiety. Similarly, Haines et al. (8) found that increased phobic anxiety scores, when controlling for other cardiovascular risk factors, were related to an increased risk of fatal ischemic heart disease during a six-year follow-up.

Evidence for increased mortality and CHD among patients with panic disorder has led to speculation regarding a variety of pathophysiological mechanisms. Based on data suggesting a high prevalence of mitral valve prolapse (MVP) among patients with panic disorder, Goldberg (9) suggested that increased cardiovascular mortality in this

population may be due to arrhythmias that occasionally accompany MVP. Decreased heart rate variability and panic-related hyperventilation precipitating coronary spasm or myocardial infarction in patients with panic disorder may also be associated with increased incidence of sudden cardiac death among these patients (5,10). However, these factors best account for cases of sudden death among patients with panic disorder and do not readily explain CHD-related findings.

Exercise, Fitness, and Panic Disorder

Increased behavioral cardiovascular risk factors, such as poor diet, alcohol abuse, and smoking may also account for the CHD-panic disorder link (1). Decreased exercise participation is one other distal factor that may be associated with increased risk for CHD in patients with panic disorder. For example, the association between heart rate variability, phobic anxiety (7), and panic disorder (11,12,13) and between heart rate variability and physical fitness (14,15) suggests that decreased heart rate variability in patients with panic disorder may be related to poorer physical fitness due to decreased exercise participation. Consistent with this, one recent study has reported avoidance of aerobic exercise in patients with panic disorder (16).

Although research is limited, several studies have examined the relationship between panic disorder, cardiovascular fitness, and exercise (17 - 20). Unfortunately, findings across studies have been contradictory. For example, Stein et al. (20) found no differences in exercise-induced anxiety symptoms between anxiety patients and nonclinical controls whereas Cameron and Hudson (17) found that many patients with panic disorder were characterized as "exercise sensitive" (i.e., reporting severe anxiety to exercise). Cardiovascular response to exercise has also been inconsistent. Two studies

found that patients with panic disorder, compared to controls, exhibited significantly greater heart rate reactivity during exercise despite similar resting heart rates (18,19). However, Stein et al. (20) reported higher baseline heart rates in patients with panic disorder compared to controls and similar heart rate reactivity (as well as similar biochemical response) during exercise.

Evaluation of cardiovascular fitness has also yielded equivocal findings with some data suggesting that patients with panic disorder exhibit lower levels of fitness and decreased exercise tolerance (e.g., discontinuing submaximal exercise testing) relative to nonclinical controls (16,19,20) but others indicating no differences in exercise tolerance or peak treadmill performance (18). In sum, findings regarding the relationship between exercise, fitness, and panic disorder are inconclusive and tend to be contradictory along a number of lines.

Cognitive Mediation and Moderation of Exercise

Cognitive variables may help elucidate the link between panic disorder and CHD and may also account for some of the previous conflicting findings. Anxiety sensitivity is a cognitive variable that may be associated with decreased fitness and exercise intolerance in panic disorder. Anxiety sensitivity refers to fear of autonomic arousal based on the belief that certain symptoms have threatening consequences. For example, an individual with high anxiety sensitivity may believe that a rapid heart rate indicates an impending heart attack whereas someone with low anxiety sensitivity would perceive a rapid heart rate as merely the normal result of physical exertion. Patients with panic disorder generally exhibit high levels of anxiety sensitivity (21). Despite this general tendency toward high anxiety sensitivity, patients vary in terms of the relative level and

type of fears (22). For some, high anxiety sensitivity scores may be due to cardiac-specific fears (e.g., "it scares me when my heart beats rapidly) (23). Cardiopulmonary fears may be responsible for exercise intolerance and physical inactivity due to catastrophic misinterpretation regarding the symptoms typically produced during exertion.

Present Study

The present study was designed to evaluate cardiovascular fitness in patients with panic disorder but, in an effort to clarify previous research in this area, different parameters believed to affect the performance of these patients during cardiovascular assessment were assessed. In other words, we evaluated whether cardiovascular fitness estimates for patients with panic disorder are accurate (i.e., reflect true fitness) or whether fitness estimates are adversely biased by individual difference variables or contextual parameters. For example, previous research has not assessed anxiety responses during fitness testing to control for the effects of anxiety on fitness estimations.

The individual difference variable of interest, anxiety sensitivity, was expected to be associated with poorer cardiovascular fitness and exercise intolerance. Moreover, anxiety sensitivity was hypothesized to mediate or moderate the relationship between panic disorder and fitness estimates. Lower $VO_{2\ max}$ estimates were hypothesized to reflect both true fitness status as well as a systematic measurement bias that underestimates fitness. In this regard, individuals who exhibit high levels of anxiety sensitivity (e.g., patients with panic disorder) were expected to exhibit poor performance on a submaximal cycle ergometer test for a number of reasons: (1) poorer fitness from chronic exercise avoidance (valid fitness component), (2) excessive heart rate reactivity

due to exercise induced anxiety (systematic measurement bias), and (3) premature termination due to fear of exercise-induced arousal (systematic measurement bias).

A contextual factor, heart rate perception, was manipulated to determine its effect on fitness estimation. Patients with panic disorder exhibit a bias for attending to bodily sensations, suggesting that they should be more reactive to cardiac feedback than nonclinical controls (24). In the context of ergometer testing, focusing on heart rate may lead to anxiety and poorer performance for individuals with panic disorder or high anxiety sensitivity. It was hypothesized that: (1) patients with panic disorder would show poorer exercise tolerance and cardiovascular fitness relative to nonclinical controls, and (2) heart rate feedback would create poorer performance for patients with cardiovascular fears.

Method

Participants

The sample consisted of 54 participants (27 panic disorder, 27 nonclinical control) meeting the following entry criteria: (a) no evidence of current or past schizophrenia or organic mental disorder, (b) no evidence of serious suicide intent, (c) no current use of beta blockers or other medications that significantly change heart rate response to exercise, (d) no significant medical history of respiratory disease, renal disease, heart disease, epilepsy, or stroke, and (e) no history of smoking. Clinical participants were drawn from a pool of patients presenting at an academic research center specializing in the evaluation and treatment of anxiety disorders. Nonclinical volunteers were solicited through advertisement in a newsletter.

Diagnostic assessment was based on a structured diagnostic interview using the Structured Clinical Interview for DSM-IV (SCID; 25). Interviews were conducted by advanced graduate students in clinical psychology who had received extensive training in SCID administration and scoring. Each interview was reviewed by a licensed clinical psychologist during weekly staff meetings. Randomly selected interviews from the lab have been found to show very high interrater reliability for panic disorder and all Axis I conditions (26). Medication status and medical history were assessed based on a semi-structured clinical interview and in consultation with the project physician. After complete description of the study, written informed consent was obtained.

Design

The study employed a 2 X 2 factorial design with diagnostic status (panic disorder versus nonclinical control) and heart rate feedback (feedback versus no feedback) as between subject factors. Subjects were randomly assigned to Feedback or No Feedback conditions (see Procedure for a description).

Assessments

Self-Report Measures

Anxiety Sensitivity Index (ASI). The ASI (27) is a 16 item self-report measure of the fear of bodily sensations associated with arousal. Each item consists of a possible negative consequence of experiencing anxiety symptoms. The ASI has demonstrated high internal consistency and satisfactory test-retest reliability (28).

Acute Panic Inventory (API). The API is a 17-item inventory for assessing symptoms of arousal associated with panic attacks (29). A subjective units of distress (SUDS) rating was added to assess subjective anxiety (0 - No Anxiety at All, 100 - The

Worst Anxiety Imaginable). Consistent with recommendations from the scale authors, as well as DSM-IV criteria, the presence of a panic attack was based on four or more symptoms rated moderate to severe along with a 30 point rise in SUDS level from baseline.

Psychophysiological Measures

$VO_{2\ max}$. Heart rate was continuously monitored using a Polar Electro wireless ECG monitor. The device consists of a heart rate sensor that is fitted around the chest and a small remote display. True maximal oxygen uptake ($VO_{2\ max}$) is estimated using an equation that combines heart rate, stroke volume, and arterial and venous oxygen levels [$VO_2 = HR \times SV \times (C_A O_2 - C_V O_2)$]. In this study, $VO_{2\ max}$ was estimated using a Monark 818E Cycle Ergometer, and using the US Air Force cycle ergometer protocol (30). This estimate is based on heart rate reactivity levels corresponding to a projected work load (determined by age, height, weight, and sex).

Exercise Tolerance. Exercise tolerance was indexed as premature discontinuation of the cycle ergometer protocol (see description below).

Procedure

Following written informed consent, participants completed the screening interview, SCID, and an assessment battery consisting of the self-report measures. Height and weight was measured using a balance beam scale. Participants were seated on a stationary bicycle for a five minute resting baseline followed by the cycle ergometer test. A post-test API was completed immediately following the test to assess for symptoms and subjective anxiety during the test.

Cycle Ergometer Test

Participants were given general instructions for the cycle ergometer test and specific instructions depending on the heart rate feedback condition. Participants were fitted with the heart rate monitor and sat at rest on the cycle to establish a baseline heart rate. During this time, the experimenter entered the participant's personal data into the computer, including age, height, weight, and sex. The participant was instructed to begin pedaling at the same rate as an audible metronome set at 100 beats per minute, equal to a pedaling rate of 50 revolutions per minute. This rate was kept constant throughout the test and was monitored by the experimenter. The participant pedaled with increasingly difficult workloads until heart rate exceeded 125 beats per minute after pedaling at a given workload for one minute. Once this workload was reached, it remained in effect for the remainder of the test. A valid test requires six minutes at a constant work load with a heart rate range typically between 130-165 beats per minute.

General Instructions

Participants were given the following instructions: "This test will measure your general level of physical fitness and estimate your aerobic capacity. It will also help us to assess the relationship between several psychological factors and physical performance. You should pedal at exactly the same rate as this metronome."

Feedback Condition. Participants assigned to this condition were provided with continuous heart rate feedback throughout the cycle ergometer test. To ensure awareness of heart rate, participants were asked to closely monitor the heart rate display and to be prepared to read the heart rate when prompted by the experimenter:

"During the test you should pay close attention to your heart rate, which is displayed on the watch in front of you. I will ask you for your heart rate periodically throughout the test."

The experimenter asked the individual for heart rate readings at baseline and at one minute intervals during the test. No other communication took place during the test.

No Feedback Condition. Participants in this condition were not provided with heart rate feedback during the test. Only the experimenter was able to monitor the heart rate display, which was placed out of the participant's view. In this condition, the experimenter read the heart rate display without providing any feedback or comments to the participant. No communication took place between the experimenter and participant for the duration of the test.

Results

Baseline Comparisons of Demographic and Clinical Characteristics

Two (Panic Disorder, Nonclinical Control) x two (Feedback, No Feedback) ANOVAs were used to evaluate differences across diagnostic groups and experimental conditions at baseline on demographic and clinical characteristics (see Table 1). There were no differences in terms of age, sex, race, or BMI across groups or conditions. As expected, patients with panic disorder exhibited higher levels of anxiety sensitivity relative to controls [$F(1,53) = 29.9$, $p < .0001$].

Effects of Diagnostic Category and HR Feedback on Ergometer Testing

Two (Panic Disorder, Nonclinical Control) x two (Feedback, No Feedback) ANOVAs assessing the effects of diagnostic group status and the heart rate manipulation on ergometer testing indicated that patients with panic disorder achieved significantly

Table 1

Baseline Comparisons on Demographic and Clinical Characteristics

	Panic Disorder		Nonclinical Control	
	HRF (n = 12)	No HRF (n =15)	HRF (n = 14)	No HRF (n =13)
Sex				
% Female	41[a]	53[a]	50[a]	46[a]
n	5/12	8/15	7/14	6/13
Race				
% Caucasian	92[a]	87[a]	86[a]	77[a]
n	11/12	13/15	12/14	10/13
Age (years)				
Mean	35.6 [a]	38.7 [a]	34.8[a]	36.0[a]
SD	11.3	11.3	10.6	11.9
Body Mass Index (BMI)				
Mean	24.1[a]	25.5[a]	24.6[a]	25.2[a]
SD	4.4	4.3	4.0	4.3
Anxiety Sensitivity (ASI)				
Mean	30.4[a]	27.8[a]	12.8[b]	14.2[b]
SD	10.3	9.6	9.7	9.5

Note. Means with different superscripts differ significantly ($p < .05$).

HRF: Heart Rate Feedback condition, No HRF: No Heart Rate Feedback condition, ASI = Anxiety Sensitivity Index.

lower $VO_{2\ max}$ estimations [$F(1,53) = 7.20$, $p < .01$] but there was no effect for condition and no interaction effect. In order to evaluate whether anxiety significantly impacted these group differences, the analysis was rerun covarying maximum anxiety during the

ergometer protocol. After controlling for anxiety, diagnosis still significantly predicted $VO_{2\ max}$ [$F(1,52) = 7.17$, $p < .01$]. Patients showed significantly elevated heart rates prior to the test [$F(1,53) = 5.81$, $p < .05$] but both groups achieved comparable maximum heart rates during the test [$F(1,53) = 0.88$, $p > .05$]. Interestingly, subjective anxiety was not significantly related to baseline HR ($r = .16$, $p > .05$) suggesting that elevated resting heart rates are not simply attributable to anticipatory anxiety (baseline SUDS and HR were also not significantly associated when evaluating patients and controls separately).

Logistic regression revealed a trend for patients with panic disorder to discontinue the test more often than nonclinical controls [$\chi^2(1, N = 54) = 3.24$, $p = .07$, Odds Ratio = 2.7, Confidence Interval: 0.86-9.4] with 33.3% of patients and 14.3% of controls discontinuing the test prematurely. There were no significant differences in subjective anxiety for completers relative to those discontinuing the test. Condition and the condition by diagnostic group interaction did not predict exercise tolerance.

Symptoms and SUDS means and standard deviations across conditions are presented in Table 2. Patients with panic disorder showed higher baseline API [$F(1,53) = 7.17$, $p < .01$] and SUDS [$F(1,53) = 15.93$, $p < .001$] ratings. Separate analyses of covariance were used to assess diagnostic group differences at post-test, controlling for baseline levels. These analyses indicated no significant differences indicating similar subjective reactivity to exercise after controlling for baseline levels (see Table 2). There were also no condition main effects or diagnosis by condition interactions predicting subjective responding.

Table 2

VO_2 Max Estimates, Cardiovascular and Subjective Reactivity During the Cycle Ergometry Test for Patients with Panic Disorder and Nonclinical Controls across Heart Rate Feedback Conditions

	PD-HRF (n=12)		PD-No HRF (n=15)		NC-HRF (n=14)		NC-No HRF (n=13)		
	M	SD	M	SD	M	SD	M	SD	Significant Findi
VO_2 Max	26.3	11.3	26.5	11.4	32.4	9.1	35.7	10.5	PD>NC
Heart Rate									
Pre-Cycle	86.1	14.0	84.7	13.2	80.1	13.6	79.5	13.9	PD>NC
Max-Cycle	162.8	14.9	159.5	14.6	157.0	13.6	152.5	13.8	
Anxiety (SUDS)									
Pre-Cycle	21.6	16.4	17.3	16.6	4.5	15.9	5.5	16.0	PD>NC
Max-Cycle	29.2	23.3	19.3	21.5	11.4	22.5	11.5	23.0	
Symptoms (API)									
Pre-Cycle	3.5	3.7	2.9	3.6	1.6	3.5	0.8	3.6	PD>NC
Max-Cycle	10.6	6.5	12.7	6.8	7.8	3.6	5.8	3.6	

Note. PD = Panic Disorder, NC = Nonclinical Controls, HRF = Heart Rate Feedback Condition, No HRF = No Heart Rate Feedback Condition.

*Post analyses controlled for Pre-cycle levels, all were non-significant.

Consistent with other reports, relatively few patients with panic disorder (2/27, 7%) experienced panic attacks during the test. Both individuals were in the HR feedback condition. None of the control participants experienced panic during the protocol.

Effects of Anxiety Sensitivity and HR Feedback on Ergometer Testing

Aside from diagnostic status, a similar set of analyses was used to examine the effects of anxiety sensitivity (AS) scores and HR feedback on physiological and subjective responding to the ergometer test. A stepwise regression procedure was used which simultaneously entered the main effect predictors (AS scores, HR feedback condition) as the first step with the interaction term entered as the second step. These analyses revealed no significant main effects for any of the outcome measures. However, the AS x Condition interaction significantly predicted $VO_{2\ max}$ [R^2 change = .16, $F(1,51) = 9.66$, $p < .01$].

To determine whether the form of the interaction conformed to prediction, specific high and low values for the ASI and HR feedback condition (i.e., feedback, no feedback) were inserted in the regression equation used to predict $VO_{2\ max}$ (31). The high and low values for the ASI were one standard deviation above and below the mean. The interaction was consistent with prediction (see Figure 1) in that $VO_{2\ max}$ levels were lower in high anxiety sensitivity individuals when HR feedback was provided but feedback did not significantly affect $VO_{2\ max}$ estimates for low AS subjects. AS, HR feedback, and their interaction did not predict exercise tolerance. Although AS was highly associated with higher levels of symptoms [$F(1,53) = 12.9$, $p < .001$, standardized $\beta = .43$] and SUDS [$F(1,53) = 39.4$, $p < .0001$, standardized $\beta = .61$] at baseline, AS, condition, and the AS x HR feedback interaction did not predict subjective responding to the challenge.

Figure 1

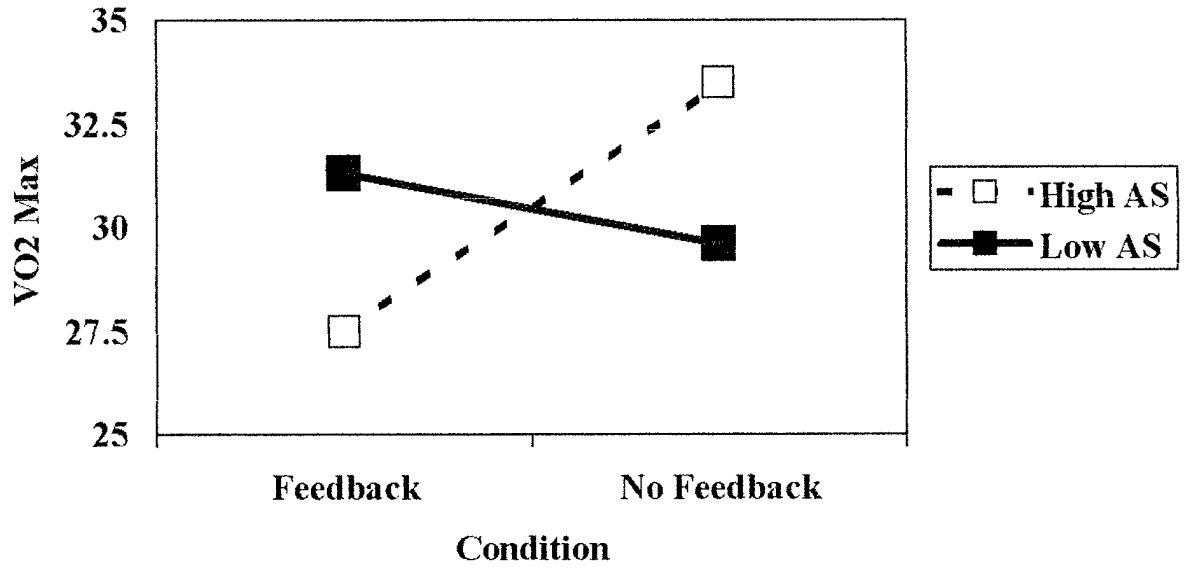

Figure 1. Interactive effects of Anxiety Sensitivity (AS) and Heart Rate (HR) Feedback on $VO_{2\ max}$ estimations. High AS individuals perform more poorly in the context of HR feedback compared to high AS individuals receiving no HR feedback.

Moderator/Mediator Analyses of $VO_{2\ max}$

The prediction that anxiety sensitivity would mediate or moderate the relationship between diagnostic status and fitness estimation was evaluated using analytic strategies outlined by Baron and Kenny (32). Mediation analyses, involving the simultaneous prediction of $VO_{2\ max}$ by diagnostic status and anxiety sensitivity (controlling for condition), indicated that both variables were independent predictors of fitness [Diagnostic Status: $t(53) = -3.17$, $p < .01$, standardized $b = -.45$, AS: $t(53) = -2.07$, $p < .05$, standardized $\beta = -.28$]. When controlling for SUDS during the test, only diagnosis

remained as a significant predictor [Diagnostic Status: $t(52) = -3.03$, $p < .01$, standardized $\beta = -.45$]. Moderator analyses, evaluating the interaction between diagnostic status and level of anxiety sensitivity controlling for condition, indicated a significant interaction term [$t(53) = 2.60$, $p < .05$, standardized $\beta = .63$]. The form of this interaction was analyzed in the manner described above and indicated that patients with relatively higher AS levels performed more poorly relative to patients with relatively lower AS levels whereas AS level did not impact fitness estimations for nonclinical participants. In sum, these analyses suggest that levels of AS moderate the impact of diagnostic status on estimation of fitness.

Evaluation of Medication Effects

Forty-eight percent of the patients were taking medications and no controls were taking psychotropic medication [benzodiazepines alone ($n = 4$), antidepressants alone ($n = 2$), both antidepressants and benzodiazepines ($n = 7$)]. Medication status (yes, no) did not differ across condition for patients. Pre-challenge benzodiazepine doses have been found to reduce fearful responding to biological challenge (33). In the present study, participants were asked to refrain from their regularly scheduled dose prior to the ergometer test. In order to determine whether medication status confounded responding on either challenge task, analyses were rerun controlling for the effects of medication status (medication, no medication). Medication status was not associated with subjective response or physiological response to the test.

Discussion

The validity of exercise testing in the presence of anxiety has been questioned because anxiety interferes with accurate cardiovascular measurement (34). The focus of

present experiment was to evaluate fitness estimations for patients with panic disorder. Moreover, we attempted to disentangle the contributions of actual fitness levels from several hypothesized biasing factors, such as anxiety, that were expected to affect fitness estimations.

Generally, hypotheses were supported. The effect of diagnosis on $VO_{2\ max}$ estimates is consistent with other reports (16,19,20) indicating that patients with panic disorder possess poorer cardiovascular fitness but the fact that this effect remains after controlling for anxiety levels indicates that this estimation is likely to be based on actual fitness levels and does not simply result from anxiety reactions during the test. Higher tonic cardiovascular levels in patients is also consistent with other reports (20) and, in this particular study it appears that elevated heart rate was not due to anticipatory anxiety. These data provide evidence for a link between panic disorder and eventual development of CHD.

Importantly, two interaction effects emerged indicating that, despite evidence for poorer fitness in patients with panic disorder, fitness estimates will be biased when several factors co-occur. First, anxiety sensitivity appears to contribute to a systematic underestimation of fitness. The anxiety sensitivity - heart rate feedback interaction indicates that individuals with this cognitive disposition may perform more poorly when they are provided with heart rate feedback during exercise. The anxiety sensitivity - diagnosis interaction shows essentially the same pattern of findings but emphasizes that this cognitive disposition will have a more dramatic affect among patients with panic disorder and relatively little effect on nonclinical samples in the context of fitness testing. Unless accounted for, these biases will lead to underestimation of fitness in patients with

panic disorder (at least in terms of submaximal fitness testing). It is notable that data from a study underway suggests that anxiety sensitivity also affects fitness estimations during maximal fitness testing (35).

It is important to recognize that anxiety sensitivity is not isomorphic with a panic disorder diagnosis. High levels of anxiety sensitivity are commonly seen in panic disorder but there are individual differences in the manner in which anxiety sensitivity manifests itself in these patients (22). In general, it appears that anxiety sensitivity affects fitness estimates but not actual fitness levels. Mediation analyses indicate that anxiety sensitivity independently predicts fitness estimations after controlling for the effects of diagnostic status. This relationship disappears, however, when controlling for subjective anxiety (whereas the relationship between diagnostic status and fitness is not affected when controlling for anxiety) suggesting that anxiety sensitivity will bias fitness because of its relationship with anxiety. On the other hand, there was no data to support our hypothesis that anxiety sensitivity would also be associated with actual fitness levels.

Only two patients with panic disorder (2/27 = 3%) panicked in response to the ergometer test suggesting that submaximal exercise is minimally panicogenic. This result is similar to earlier studies in which few individuals panicked in response to exercise (20). In contrast, biological challenge tests have produced strikingly different results. For example, lactate infusion causes panic in a substantial percentage of patients with panic disorder (36). Exercise is known to increase lactate levels in the body, and this lactate increase is substantially greater than lactate levels achieved through sodium lactate challenges (37). Thus, it might be expected that a greater percentage of patients would exhibit significant increases in anxiety or panic attacks during the ergometer test.

The differential panicogenicity of lactate challenge versus exercise testing is likely to be due to expectancy differences inherent to the procedures such that exercise-induced sensations are perceived to be predictable and controllable relative to lactate-induced sensations.

In general, findings are consistent with the idea that poor cardiovascular conditioning may contribute to increased cardiovascular mortality in patients with panic disorder. Additional research including longitudinal analyses of risk factors over time is required to elucidate the relationship between panic disorder and CHD. There is also some suggestion that a subgroup of panic disordered individuals with high anxiety sensitivity may avoid high intensity exercise sensations due to preexisting fears of cardiovascular sensations. This subgroup of patients may be at greater risk for CHD.

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Discussion

The Current Studies

Expectancy theory posits that anxiety sensitivity may serve as a premorbid risk factro for the development of anxiety pathology (Reiss & McNally, 1985; Reiss, 1991). In conjunction with Maller and Reiss' (1992) prospective study, these data provide strong evidence for this assertion. In addition, the effects of anxiety sensitivity appear to be specific to anxiety pathology as opposed to general psychopathology (Schmidt, Lerew, & Joiner, 1998). Anxiety sensitivity however, appears to account for a relatively small proportion of the variance in pathology. Other vulnerability factors such as body vigilance also appear to present cognitive diatheses which increase the chances of developing not only anxiety pathology but also of experiencing increased physical and mental distress and functional impairment (Schmidt, Lerew, & Jackson, 1997; Schmidt & Lerew, 1998).

The Scope of Relevant Effects

Psychological Impairment

Analysis of the USAFA ASI data in terms of more extreme values provides what is possibly the most telling picture of the effects of AS. Schmidt, Lerew, and Jackson (1997) reported that those individuals scoring in the highest quartile on the ASI were at almost twice the risk for panic in comparison to the rest of the sample. Forty-five percent of this highest quartile ASI group scored at or above the "moderate" level of clinically significant anxiety on the BAI. Of this high AS group, seven percent reported multiple panic attacks with panic-related worry and behavioral impairment. Some individuals reporting these additional symptoms would have likely met diagnostic criteria for panic

disorder. Furthermore, those who experienced a panic attack during BCT reported higher levels of depression and hopelessness. Another method of guaging the impact of elevated anxiety sensitivity was to measure disability as indexed by visits to peer counselors. Axiety sensitivity was associated with the number of visits to peer counselors indicating that high AS individuals may have been in greater need of support. Interestingly, AS did not predict visits to the counseling center, a more formal mental health treatment approach. This may be best understood in the context of BCT, in which visits with peer counselors are more easily accessible, more discreet, and less likely to result in "negative" outcome, such as a formal psychiatric diagnosis.

The single laboratory-based study presented here (Schmidt et al., under review) yielded comparatively little evidence for AS-related subjective distress. During the cycle ergometer test, only two subjects experienced clinically significant levels of subjective anxiety. However, this is not to say that subjective anxiety was without consequence. High AS individuals appeared to be affected negatively in that they experienced greater heart rate increases, likely contributing to lower test scores. This study is discussed further below.

Functional Impairment

Aside from psychological impairment, these studies indicate that elevated AS is associated with measurable physical and behavioral consequences. For example, higher ASI scores predicted greater levels of impairment in peer relationships, supervisory relationships, and overall self-rated impairment (Schmidt & Lerew, 1998). This may be one of the most interesting findings to come out of this research. These more socially-based consequences of elevated AS illustrate the far-reaching effects that AS may exert

on individuals' lives. Also, it is unusual for studies to report (or in fact, collect data) on correlates of elevated AS aside from anxiety or panic. Of course, it may be argued that the significance of impaired relationships is probably not on such a high level of importance as are panic attacks. Nevertheless, it is intriguing to speculate as to why anxiety sensitivity, primarily a fear of bodily sensations, would be associated with social interference. One hypotheses is that for some high AS individuals social interactions may necessitate an increase in arousal, which then leads to subsequent increases in anxiety. However, the high valence of the social context in which this research was conducted (i.e., USAFA) must be considered. As a basic trainee, almost every interaction with supervisors is by definition anxiety provoking. Supervisors demand specific responses to nearly all prompts, and are highly critical of protocol violations (that are defined be either strict or relaxed criteria of the supervisor's choosing). In this case, the environment presents a clear limitation on our extrapolation of the meaning of impairment in supervisory relationships. But what is to be made of the finding that high AS individuals also reported relative impairment in their relationships with their peers (i.e., other basic trainees)? This finding may be more generalizeable to other environments (i.e., the workplace), as contextual factors are less salient here than those involved in supervisory interactions. Peers would be expected to present much less of a threat than supervisors, and in fact might be expected to provide a considerable amount of social support in the midst of basic training. With this point in mind, functional impairment in terms of peer relationships could be quite devastating in an environment where one has few sources of support aside from one's peers. It is possible that high AS in individuals reporting distress in peer relationships is due to elevtions in the social fear subfactor of the ASI as

opposed to other fears (e.g., losing mental control, physical concerns). The AS subfactors are discussed in more detail below.

In considering the functional relevancy of elevated AS, it would be helpful to guage individual performance in a variety of areas, aside from self-report, and on a relatively long-term basis. Jackson (1988) performed a cursory data analysis of follow-up data collected at a one year follow-up of USAFA cadets. This data included ratings of academic performance (grade point average; GPA), military performance (military performance average; MPA), and physical performance (physical evaluation average; PEA). After controlling for trait anxiety, AS significantly predicted PEA [$F(2,870) = 5.30$, $p < .01$] and approached significance in predicting GPA [$F(2,870) = 2.77$, $p = .06$] at one year follow-up. In both cases, higher AS was associated with poorer performance. This initial look at performance follow-up data is promising, and suggests that the effects of elevated AS may be pervasive and long-lasting. Additional analyses will help to explore these effects.

Health Issues

The pervasive effects of higher levels of AS were also shown to produce higher levels of physical health impairment (Schmidt, Lerew, & Jackson, 1997; Schmidt & Lerew, 1998; Schmidt et al., under review). For example, in the USAFA population, high AS was predictive of increased number of visits to the health clinic and number of days on sick call. Those participants in the upper quartile in terms of ASI scores reportedly spent 50 percent more time at sick call than others lower in AS (Schmidt, Lerew, & Jackson, 1997). Data reported by Schmidt et al. (under review) speaks more directly to the issue of the effects of AS on physical health. Findings suggested that AS may lead to

poorer cardiovascular fitness due to anxiety responses to exercise. If individuals with elevated AS avoid exercise, it would be expected that poorer cardiovascular fitness could be an end result. However, it appears that more proximally, AS affects cardiovascular fitness estimates rather than actual cardiovascular fitness due to elevated anxiety (and increased heart rate) during testing. In the least, this finding is important to consider when testing individuals such as those diagnosed with panic disorder, as improper conclusions about fitness may be reached erroneously.

Clinical Significance

When do ASI scores become practically important? Also, in reducing AS, what is more important--total change in AS or reduction past a certain cutoff point. "Elevated" or "High" AS has been defined as a score of approximately 20 (Donnell & McNally; 1989). However, a score of 25 on the ASI does not necessarily hold clinical implications, especially in the absence of negative life events and stressors. Likewise, as demonstrated by Schmidt, Lerew, and Jackson (1997), "low" ASI scores such as those exhibited by some USAFA cadets do not necessarily imply an absence of AS-related consequences. The question of clinical significance may be best explored in treatment outcome studies measuring AS at pre- and post-treatment in addition to other behavioral and psychological variables. Recently, Schmidt (1998) reviewed data collected during ongoing treatment of panic disorder patients in our lab. In additon to patient-rated disability, measures of anxiety symptoms, phobic avoidance, and clinician-rated level of impairment were collected. With one sample of patients treated for panic, both changes in ASI scores and absolute ASI score were predictive of all outcome measures at a three-month follow-up. Based on a median split (i.e., "high" or "low") of ASI scores after

treatment, patients with low ASI scores were approximately 13 times more likely to be recovered at follow-up. Similarly, those patients showing ASI reductions greater than 15 points were nearly 9 times more likely to be recovered at follow-up. In both cases, recovery was measured by both the clinician and the patient. These findings suggest that reductions in anxiety sensitivity as as well as its absolute level at the end of treatment predict the course of the pathology (Schmidt, 1998).

It is clear from the current research that although levels of AS may vary widely among populations (e.g., nonclinical vs. anxiety disordered) and environments (e.g.,civilian vs. military). In order to avoid the enigma of clinical significance, future research should be careful to measure both change scores and absolute levels of AS in addition to exploring hypothetical clinical cutoff scores. Some advise that in order to determine the clinical significance of change as opposed to statistical significance, researchers should require both a significant change from baseline and a reduction in AS to a level below an absolute cutoff score (Speer, 1992). Such a method will be helpful and would help to ensure that future studies are comparable despite using different populations, treatments, or conditions.

The Relative Contribution of AS

In the current studies, the association between anxiety sensitivity and outcome variables such panic attack occurrence, psychological distress, and physical and functional impairment, while significant, accounted for only a limited amount of variance suggesting that there are other important factors involved in the pathogenesis of panic. In terms of panic attack frequency for example, the unique variance accounted for by anxiety sensitivity is perhaps only 2%. In comparison, studies evaluating the relationship

between specific candidate genes and anxiety suggest that some of the individual functional polymorphisms may account for as much as 5% of the overall phenotypic variation (Lesch et al., 1996). One factor of importance regarding the large sample of data collected at USAFA is that the sample showed relatively low levels of anxiety sensitivity. This restriction in ASI score range is likely to have led to an underestimation of the variance accounted for by AS in many analyses. Considering other variables such as discomfort intolerance and body vigilance (discussed in more detail below) in addition to AS, the level of association with psychopathology increases substantially. In other words, if rather than considering only AS, we consider a "vulnerability constellation," it appears that we may be able to account for as much as 30 percent of the variance in psychopatholgy (Lerew, Schmidt, & Jackson, in press).

It may be interesting to approach a future study with this constellation perspective rather than a singular vulnerability perspective. For example, a composite measure of vulnerability could be constructed that borrows items from measures of several domains (e.g., bodily hypervigilance, the bodily fears subcomponent of the ASI, and discomfort intolerance). Such a measure which concentrates on bodily fears may be compared to another composite measure with a concentration on fears of loss of cognitive control. Perhaps differential measures would be more relevant for specific populations (e.g., underwater welders or astronauts versus public speakers) where certain traits are important in terms of screening in or screening out candidates. Another approach may be to use only the most salient items from measures of several domains and to combine them in an attempt to construct a more general measure of psychopathology vulnerability. Such a concept is not completely foreign to psychology researchers. For example, someone

with a generally negative cogntive schema (Beck, 1988) might be more likely to espouse several fears in a hypothetical vulnerability constellation.

Thus, while there is strong evidence to support the role of anxiety sensitivity in panic and other psychopathology, considerable additional work is needed to identify other psychological parameters as well as the interaction between psychological, physiological, and even genetic factors in the pathogenesis of anxiety. These factors may interact with dispositional variables such as anxiety sensitivity to predict the development of psychopathology and related disability. There is a growing literature to suggest that cognitive factors, such as perceived control, influence anxious responding in both clinical (Carter, Hollon, Carson, & Shelton, 1995; Rapee, Mattick, & Murrell, 1986; Sanderson, Rapee, & Barlow, 1989) and nonclinical (Schmidt & Telch, 1994; Telch et al., 1996) populations. Evaluation of other dispositional variables and their interactions with contextual factors is warranted to better explain the relationship between underlying psychological characteristics and the development of psychopathology.

Other Vulnerability Factors

The process of monitoring internal states has broad relevance for many theories of emotion and has been described by terms such as visceral perception, autonomic perception, symptom perception, and interoception (Ehlers & Breuer, 1992; McLeod & Hoehn-Saric, 1993; Pennebaker, 1982). Internal awareness is responsible for important health-relevant behaviors such as eating and drinking and is, therefore, critical for adaptive monitoring of physiological functioning. As illustrated by the AS research presented here, bodily sensations have taken on increased importance in psychological conceptualizations of panic and panic disorder. Cognitive models of panic (Clark, 1986;

Reiss & McNally, 1985) suggest that patients with panic disorder possess a cognitive disposition to panic in the context of aversive sensations. The perception of bodily sensations is considered a necessary step in the pathogenesis of panic. Although the perception of bodily sensations is central to psychological models of panic (Barlow, 1988; Clark, 1986), a comprehensive understanding of the role of attentional vigilance to bodily cues in panic disorder is lacking.

There are individual differences in monitoring and evaluation of internal sensations. Some individuals fail to seek medical care despite symptoms or report no awareness of significant physiological events such as myocardial infarction (Beunderman, van Dis, & Duyvis, 1987). Others closely monitor internal sensations and repeatedly present for medical evaluation when there is no evidence to suggest organic etiology. In the case of panic disorder, individuals appear to excessively monitor internal sensations because they report very high levels of symptoms and repeatedly seek out medical evaluations despite reassurances from health care providers (Weissman, 1991).

Ehlers (1993) has outlined three hypotheses to explain increased symptom perception in panic disorder. Patients with panic disorder may be more apt to perceive autonomic sensations because of: greater physiological reactivity; enhanced ability to perceive physiological sensations; or increased attention to physiological sensations. Substantial research by Ehlers and colleagues (Ehlers & Maddock, 1986; Ehlers & Breuer, 1992; Ehlers, Breuer, Dohn, & Fiegenbaum, 1995) has suggested that patients with panic disorder are more skilled in detecting bodily processes ("interoceptive acuity") such as heartbeat than are normal controls. For example, patients with panic disorder have scored higher on measures of self-reported cardiac awareness (Ehlers & Maddock,

1988), and have been shown to be better (i.e., more accurate) in perceiving their heart rate (and, in fact, perceiving a heart rate at all; Ehlers & Breuer, 1992) than nonclinical controls. However, earlier studies (Ehlers, Margraf, & Roth, 1988; Ehlers, Margraf, Roth, Taylor, & Birbaumer, 1988) found no differences between patients with panic disorder and nonclinical controls in cardiac perception.

Body Vigilance

Aside from interoceptive acuity, another variable related to awareness of bodily sensations is body vigilance (Schmidt, Lerew, & Trakowski, 1997). Body vigilance refers to conscious attention focused on internal bodily sensations and perturbations. Schmidt et al. (1997) present a description of the role of body vigilance in panic disorder. In this model, the experience of spontaneous, or "false alarm," panic attacks is necessary for the development of panic disorder. These false alarms may create worry about autonomic arousal and the perceived consequences of arousal. According to Barlow (1988), false alarms result in a shift in attentional focus from the environment to the body. The attentional shift to somatic events is a necessary consequence of the false alarm, as subsequent worry about more false alarms becomes associated with interoceptive cues. Thus, excessive body vigilance may be a natural consequence of learning to fear bodily sensations (i.e., anxiety sensitivity) and, therefore, part of the behavioral sequelae of developing panic disorder. In addition, increased attentional focus on the body should increase the likelihood of perceiving threatening interoceptive cues. For example, Pennebaker et al. (1985) found that an internal focus of attention enhances the probability of perceiving physiological changes. The increased perception of threat cues should lead

to greater fear and autonomic arousal, thus creating a vicious cycle described in the model.

The body vigilance model conforms with the increased attention hypothesis (Ehlers, 1993) which has largely been investigated by information processing paradigms evaluating preconscious biases. Information processing studies have generally indicated an enhanced attentional bias toward threat cues among patients with panic disorder (McNally, Foa, & Donnell, 1989; McNally, Riemann, & Kim, 1990). Other studies relevant to the attentional hypothesis have assessed the accuracy of attentional processes (e.g., Ehlers & Breuer, 1992). The vigilance concept is distinguished from this previous work in that it focuses on changes in conscious attention regardless of accuracy. According to this model, the act of perception, independent of its accuracy, is a process that may contribute to maintaining panic disorder.

Thus, body vigilance is described as a behavioral consequence that becomes exaggerated in the context of fear of bodily sensations. Accordingly, body vigilance should be closely linked with anxiety sensitivity (i.e., fear of autonomic arousal) but not necessarily with trait anxiety (i.e., general tendency to be fearful

The model of body vigilance described by Schmidt et al. (1997) suggests that panic-related worry (i.e., worry about autonomic arousal) should lead to vigilance for related interoceptive threat cues. This hypothesis was tested experimentally, and consistent with this model, body vigilance was significantly associated with anxiety sensitivity in both the nonclinical (r = .27) and panic disorder (r = .42) samples. The higher level of association in the clinical sample suggests that the development of a formal panic disorder syndrome may produce a closer connection between anxiety

sensitivity and body vigilance. Although body vigilance was associated with anxiety symptoms as indexed by the BAI, it was not associated with trait anxiety in the nonclinical sample indicating that body vigilance is uniquely associated with worry about arousal (i.e., anxiety sensitivity) and anxiety symptoms. It is also notable that panic attacks were generally associated with body vigilance, but panic attack frequency was not significantly associated with body vigilance or other clinical indices. Skewed panic frequency data, as well as the fact that generally low panic attack frequency may be indicative of mild symptomatology or extensive avoidance behaviors, is likely to account for this lack of association.

Assessments of anxiety sensitivity and body vigilance over time were also consistent with the relationship predicted by the body vigilance model. In the nonclinical sample, changes in anxiety symptoms and anxiety sensitivity, but not trait anxiety or depression, were positively correlated with changes in body vigilance. Data from the panic disorder sample indicated that changes in body vigilance were highly associated with changes in anxiety sensitivity. Moreover, pretreatment anxiety sensitivity was the only clinical variable that predicted posttreatment body vigilance levels. Interestingly, posttreatment findings indicated that treated patients showed less body vigilance than nonclinical samples suggesting that vigilance is readily malleable when it becomes the focus of treatment intervention.

Current conceptualizations of panic disorder, including the DSM nosologic distinctions between the diagnoses of panic disorder and agoraphobia, make phobic avoidance a typical, but not necessary, consequence of developing panic-related worry. On the other hand, we have argued, and consistent with fear of fear conceptualizations of

panic disorder, that body vigilance is a necessary behavioral component of the disorder because fear of body sensations will necessarily inspire vigilance. It may be important for body vigilance to be incorporated in the routine assessment of patients with panic disorder to provide a more comprehensive assessment of behavioral sequelae that is distinct from panic and anxiety symptoms, phobic avoidance, and anxiety sensitivity. Among nonclinical samples, body vigilance is likely to be a stable disposition that may, for some individuals, act as a risk factor for the development of anxiety pathology vis a vis increased perception of bodily perturbations. Also, one of the key assumptions of many theories relevant to interoception is that the individual has the capacity to accurately perceive internal states. Undoubtedly this assumption is responsible for the considerable amount of work focusing on the accuracy of internal perception (Ehlers, 1993; Pennebaker, 1982). The study by Schmidt et al. (1997) highlights the importance of the act of perception in the pathogenesis and maintenance of panic disorder. The act of perception (i.e., scanning or checking heart palpitations), relative to the accuracy of perception (i.e., accurate detection of heart beats), may be a more substantial contributor to anxiety pathology in panic disorder. Work is needed to distinguish the relative influences of the process of vigilance from the accuracy of perception in the generation and maintenance of anxiety.

Predictability/Controllability

Predictability and controllability of aversive events are believed to be important parameters that affect the generation of anxiety and panic (Barlow, 1988). For example, perceived control is one important component of Barlow's (1988) concept of psychological vulnerabilities, where one vulnerability is the sense that events in general

and emotions in particular are uncontrollable and unpredictable. The importance of control (i.e., Barlow, 1988) in the mediation of panic was demonstrated by Sanderson, Rapee, and Barlow (1989) in a CO_2 challenge study. In this study Sanderson et al. (1989) instructed patients with panic disorder that a dial placed nearby would allow them to control the flow of CO_2 (i.e., the intensity of sensations) during the test if a light was illuminated. The light was illuminated for half of the subjects (illusion of control group), but in reality, the dial was inoperative for all subjects. The illusion of control group, in line with the cognitive mediation hypothesis, was significantly less likely to panic and reported less catastrophic cognitions than the control group.

Barlow (1991) suggests that a perceived lack of control over potentially anxiety provoking environmental events (both internal emotions and external threats) may, like AS, be a psychological vulnerability factor acting as a diathesis for both pathological anxiety and depression. One's perception of control, in combination with stressors, other vulnerabilities, and coping strategies, is thought to determine whether or not an individual is likely to experience increased anxiety. Rapee, Craske, Brown, and Barlow (1996) have developed a measure of perceived control over anxiety-related events that may prove useful in the assessment and treatment of anxiety pathology. Unlike some measures of perceived control that measure an individual's generalized expectancy of control over events related to all areas of life, the ACQ is a 30-item questionnaire that is more relevant to the anxiety disorders because it measures perceived control over emotional events and external threats [e.g., "When I am in a stressful situation, I am able to stop myself from breathing too hard;" (rated 0- Strongly Disagree to 5- Strongly Agree)]. This measure has been shown to have good internal consistency (Cronbach's alpha = .89), test-retest

reliability, and convergent and discriminant validity (Rapee et al., 1996). Also, patients with panic disorder have shown significant changes on this measure after treatment (Rapee et al., 1996).

Recently, Zebb and Moore (1998) found that individuals low in percieved control reported significantly higher anxiety than individuals who reported moderate or high perceived control. Jackson (1998) used a measure of perceived control in addition to the ASI in a follow up study of psychological vulnerability in military cadets. It was assumed that perceptions of control and predictability would be associated with anxiety symptoms and that these parameters may interact with AS in the genesis of anxiety pathology. Findings were partly supportive of expectations. In general, the hypothesis that basic military training would be characterized by low levels of perceived control was supported. Surprisingly, perceived control was not independently associated with panic. On the other hand, expected interactions among perceived control and anxiety sensitivity where low perceived control contributed to anxiety, especially in high AS subjects. These preliminary findings suggest that perceived control may indeed play a critical role in the generation of panic, but further study of this variable is warranted. Although more research is warranted with perceived control, early studies such as these suggest that this variable may be another important factor in the constellation of vulnerability factors contributing to anxiety pathology.

Discomfort Intolerance

The AS conceptualization of fear does not imply that someone with elevated AS should experience a given sensation as qualitatively different from the same sensation as experienced by someone with low levels of AS. Rather, the concept of AS implies an

attribution of danger to ambiguous physical sensations. In terms of AS, one avoids a rapid heartbeat not so much because the rapid heartbeat per se is painful or uncomfortable, but rather because a danger expectancy has been assigned to the rapid heartbeat. Evidence in our lab suggested that another type of "sensitivity" was evident in some individuals. Psychological responses to biological challenges in conjunction with ASI scores suggested that some individuals respond negatively to bodily sensations despite low ASI scores. In addition, certain individuals may avoid behaviors (e.g., exercise) associated with salient bodily sensations, but may not endorse fears of these sensations. Do some individuals simply dislike and, in turn, avoid discomfort without necessarily experiencing a significant emotional reaction? To test this possibility, our lab developed the Discomfort Intolerance Scale, a short (6-item) measure designed to assess the ability to tolerate discomfort as well as avoidance of discomfort (Schmidt, 1998). Schmidt and Lerew (1998) found some evidence that individuals scoring high on this measure were more likely to experience occupational and health-related distress. Specifically, high levels of discomfort intolerance were related to poorer physical health and greater overall impairment during basic military training. In addition, and as might have been expected intuitively, increased discomfort intolerance was predictive of increased utilization of health care resources and missed work due to illness.

Is discomfort intolerance a risk factor for pathology? Preliminary findings have not suggested that it predicts mood or anxiety pathology per se, but do indicate that it may have some significant effects on health-related behaviors and on occupational impairment. It is tempting to extrapolate such findings to suggest that individuals with high levels of discomfort intolerance may be at increased risk for negative outcomes in

general, and by proxy be at risk for associated negative mood or anxiety outcomes. While the prospect of identifying yet another "vulnerability" factor is exciting, such conclusions should be tempered at this time. One problem with beginning research on discomfort intolerance is that no underlying theoretical background has been identified to support the variable. Is discomfort intolerance a specific surface trait that stems from a higher-order dimension, or is it an important trait that lies at a higher level that anxiety sensitivity? Arguments could be made for either side. A pessimestic view of discomfort intolerance may be that even if it were to be "discovered" as an important vulnerability factor for negative behavioral and mood outcomes, it may be difficult to lower levels of the trait. On the other hand, perhaps lowering the trait, if it truly exists, could be as simple as repeated exposure to discomfort in a variety of forms. These and other questions remain to be answered. For now, discomfort intolerance can be easily explored in further studies, as the DIS is easy to administer and adds little time to that often required to complete self-report inventories. Comparisons and contrasts with other factors such as AS and body vigilance will provide useful and more meaningful data in the future.

More on The Relationship between Vulnerability and Impairment

One interesting consideration is whether AS and other vulnerability factors exert their effects directly or via their relationship to mood (i.e., anxiety and/or depression) pathology. It appears that both may be true depending on the effect in question. Schmidt et al. (under review), in their study of AS and cardiovascular fitness, found that in nonclinical samples AS was relatively unimportant in fitness estimations, but for patients with panic disorder, AS had a more dramatic effect whereby elevated AS negatively affected fitness estimates. The finding of poorer cardiovascular fitness in patients with

panic disorder disappeared after controlling for anxiety levels, suggesting that subjective anxiety, an indirect effect of elevated AS, may have been to blame. Because elevated AS was not implicated in poorer fitness in the nonclinical sample, it is unrealistic to conclude that the elevated AS of the panic disorder patients was solely responsible.

In contrast, each of the psychological risk factors evaluated by Schmidt and Lerew (1988) predicted the development of impairment after controlling for other relevant variables. Many of these relationships persisted even after controlling for the effects of distress symptoms, suggesting that these psychological risk factors directly impact impairment in addition to impacting impairment vis a vis the development of anxiety and depression symptoms. In terms of the health-related outcomes such as health care utilization, we speculated that the presence of these characteristics would lead to an increased likelihood of health-seeking behaviors due to exaggerated concerns about physical and mental well-being. Many individuals with such exaggerated concerns are likely to seek out assistance without any clear awareness of significant anxiety or depression symptoms. Unfortunately, more direct measures of physical health (e.g., estimated cardiovascular fitness, immune functioning) were not possible. Such measures may however, be possible in future laboratory-based studies with fewer and more accessible subjects.

Another alternative view is that psychological variables such as AS may tap into underlying biological vulnerabilities that make individuals more prone to negative outcome (e.g., psychopathology, healtcare utilization). For example, Lesch et al. (1996) reported the identification of a specific gene related to anxiety that affects the brain's use of the neurotransmitter 5-hydroxytryptamine (serotonin). Evidence indicated that a short

version of this gene produces fewer serotonin transporter molecules, resulting in lower levels of serotonin uptake. Although the gene was found to account for only four percent of the variance in neuroticism, this finding is promising in the ongoing search for biological contributors to psychopathology. It will be interesting as future researchers combine genetic investigation tecnology with measures of psychological vulnerability in further attempts to elucidate the complicated nature of biological and psychological interactions.

Limitations and Generalizability Issues

The Sample Population

It is important to recognize that although the current series of papers approaches the subject of anxiety sensitivity and psychological vulnerability from somewhat different perspectives (e.g., psychopathology, occupational rehabilitation, vulnerability), these studies (save Schmidt, Lerew, Santiago, Trakowski, & Staab, under review) are derived from the same sample of individuals. The majority of the findings presented in the current paper were derived from a single population sampling at the United States Air Force Academy (USAFA). The United States Air Force Academy provided an opportunity to evaluate incoming first year students during their initial (highly stressful) five weeks of training at the academy, an excellent population and setting for investigation of the diathesis-stress model of psychopathology. The unique nature of this population provides several advantages for research, including accessibility and ease of monitoring. In addition, several assumptions can be made with relative confidence, including the assumption that the majority of study participants had no preexisting psychological or medical diagnoses (aside from those medical diagnoses that are eligible

for waiver, such as mild allergies or asthma). The lengthy medical screening process, stringent physical fitness requirements, and background checks inherent in the USAFA application process help to ensure a high level of health.

Unfortunately the unique, and largely homogenous, nature of this population also presents what may be the most serious limitation on the current studies in terms of generalizability. Two specific and important areas of generalizability limitations stem from: a) restriction of range in terms of outcome scores (e.g., anxiety sensitivity (ASI), anxiety (BAI), and depression (BDI) scores, and b) demographic homogeneity in terms of race and gender. The primarily self-report nature of data collection presents another important limitation.

Restriction of Range

Interestingly, cadets generally showed very low levels of anxiety sensitivity. Even the group experiencing panic attacks reported levels of anxiety sensitivity well below the average for nonclinical samples. Initial levels of anxiety (e.g., BAI; $\underline{M} = 7.3$) and depression (e.g., BDI; $\underline{M} = 6.4$) reported by Schmidt, Lerew, and Jackson (1997), despite being "elevated" in comparison to post-BCT levels, still fall well within mild to minimal ranges. Conclusions should be tempered with such restriction of range in mind. Not only are statistical results affected by range restriction, but practical meaning of change can be skewed as well. For instance, a change in BDI depression score of -8 may be considered significant in clinical practice for a patient whose score drops from 20 to 12, and thus from a "moderate" to "mild" classification. But what is to be concluded of a drop in BDI score from 9 to 1? Clearly, both scores are indicative of "mild" to "minimal" depression, and may warrant little clinical concern. Similar arguments may be made

regarding changes in ASI score occurring within the low end of the ASI score range. Several discussions in the current papers refer to the development of psychopathology in this population. Indeed, should clinical distress or impairment in social or occupational functioning be a factor, "psychopathology" is an appropriate term. However, it may be more appropriate in many cases to refer to scores in this population as "psychological symptomatology" rather than "psychopathology".

Demographic Homogeneity

The largely Caucasian and male nature of the USAFA population pose important limitations on the generalizability of findings. In the current studies, no significant findings emerged regarding ethnic differences in key variables of interest, including anxiety symptomatology (e.g., panic attacks) and anxiety sensitivity. As reported by Schmidt and Lerew (1998), race was not significantly associated with any index. This finding is positive in regard to the lack of evidence of differential effects of stress, and/or proposed vulnerability factors within the environment of basic training and USAFA. Differential ethnic findings could be reason for alarm, should they indicate significantly higher levels of symptomatology in any one ethnic group. However, when applied beyond the unique context of the Academy, the present findings are once again limited in scope. With a 77% Caucasian majority, the lack of findings regarding ethnic differences in this sample is not surprising. Statistical significance when comparing ethnic groups however, may be greatly affected by the extreme numerical differences (i.e., number of individuals) between groups. To date, the study of racial differences in the presentation of psychopathology has been relatively overlooked. The areas of panic, anxiety, and anxiety sensitivity are no exception. Extensions or replications of the studies discussed here

would be improved by efforts to oversample minority populations in an effort to more clearly elucidate racial differences. Although the ASI has been translated into several different languages, no published studies exist that compare racial groups in terms of anxiety sensitivity or in terms of ASI response patterns. These studies are warranted and could provide exciting findings.

Several interesting findings were reported in the current studies regarding gender differences, and particularly regarding gender-AS interactions. Gender was evaluated as a moderator variable because of the hypothesis that vulnerability factors would act differentially in males and females. For example, in Schmidt and Lerew (1998), the prediction of visits to the counseling center showed that greater numbers of females reported such a visit and that there was a significant sex x ASI interaction. Evaluation of the form of the interaction indicated that high anxiety sensitivity potentiated counseling center visits only for females. It appears that these risk variables may potentiate impairment among females particularly in terms of mental health outcomes. This would suggest that females may differentially interpret stress or their stress reactions. Our lab and others have found gender differences in measures of appraisal that indicate potential differences in particular thinking errors (e.g., catastrophizing, overestimation of threat likelihood). For example, Stewart, Taylor and Baker (1997) recently found gender differences in anxiety sensitivity in a nonclinical sample of college students. Further investigation is needed to determine the specific nature of these gender differences and the factors that lead to potentiation of impairment in females.

Females appear to be at greater risk for the development of panic disorder, and are particularly at risk for the development of panic disorder with agoraphobia (Schmidt,

Lerew, & Koselka, 1996). Traditionally feminine gender roles, anxiety sensitivity, and panic-related appraisals may account for the differentially higher rates of panic disorder among females. Data from USAFA suggest that adaptation to stress may be another variable that could account for gender differences in anxiety. Stressors are ubiquitous and individual responses to stressors are diverse. Most individuals adapt to typical stressors but, individual vulnerabilities and stressor severity together predict adaptation to the stress. For example, empirical studies have demonstrated that gender is a general risk factor, as females show greater reactivity to stressors (Baum & Grunberg, 1991) though the consequences of this greater reactivity is not yet clear.

Unfortunately, our overall understanding of gender differences in anxiety is limited. If gender is a risk factor for the development of anxiety pathology, what are the mechanisms that account for this? Biological, psychological, and sociocultural theories have been put forward, but these theories largely remain untested. The current studies suggest that one psychological construct, anxiety sensitivity, may mediate gender differences in anxiety. However, further work is needed to develop a clear understanding of the variables that underlie these gender differences. Like findings regarding ethnic differences, the gender findings reported in the current studies must be interpreted with the unique nature of the sample in mind.

The following recommendations are made for future research:

(1) More comprehensive evaluation of the singular and combined effects of biochemical, genetic, and psychological factors that may contribute to gender and/or racial differences in the development of psychopathology;

(2) Evaluation of gender and racial differences with regard to cognitive factors including anxiety sensitivity, perceived vulnerability, and other anxiety-related appraisals;

(3) Evaluation of gender and racial differences, and factors that may moderate or mediate gender and racial differences, in psychological interventions.

Self-Report

The assessment of cognitions has been described as "integral to cognitive theory and practice" (Clark, 1988, p.2). One of the main limitations of the present studies is their reliance on self-report measures for the assessment of panic and anxiety symptoms. Previous work with nonclinical samples suggests that self-report indices may lead to an overreporting of panic attacks (Wilson et al., 1992). Wilson et al. found that relatively few individuals endorsing panic attacks on a self-report measure met diagnostic criteria for "clinical" panic. In addition, the items contained in self-report questionnaires are prototypical statements, and do not necessarily match those cognitions or beliefs of individual study participants (Glass & Arnkoff, 1997). The typically understood limits of self-report measures may even be amplified in the current (i.e., USAFA) population due to underreporting of symptoms. Although some effort was made to screen unreliable reports, it is conceivable that this population, being fearful in general of negative evaluation and ever vigilant of threats to career, would tend to underreport much of their symptomatology. Clearly, a good understanding of the nature of the population in these studies is helpful, and perhaps recommended, when interpreting overall results and making conclusions.

A Broader Perspective

Expectancy Theory

Almost a decade ago, Reiss' (1991) expectancy theory laid the groundwork for the abundance of research that has since been conducted on AS and its role in clinical and nonclinical anxiety. The theory as a whole however, did not concern itself with AS alone but proposed other sensitivities and expectancies that supposedly work in conjunction with one another to determine the human fear response. Without a doubt, AS has received the bulk of the research attention to date. Nor did Reiss' original work set forth only one hypothesis (AS is a risk factor for anxiety pathology). Two questions then, deserve mention at this juncture. First, how have Reiss' other hypotheses withstood time and scrutiny, and second, what has become of the other components of expectancy theory?

Additional Hypotheses of Expectancy Theory

Hypothesis: "AS is an individual difference variable that is measured by the ASI"

An impressive array of research has been conducted to establish the ASI as a measure of of an individual variable measuring the fear of anxiety symptoms. Substantial research supports its validity, it is characterized by a high degree of internal consistency (Peterson & Reiss, 1992; McNally, 1997), and the ASI has satisfactory test-retest reliability (Reiss et al, 1986; Maller & Reiss, 1992). The ASI has been translated into German, Greek, Dutch, Mandarin, Spanish, Farsi, Frency, Catalan, and Hebrew (Peterson & Reiss, 1992), and research on a childhood version indicates that it has similar properties to the original (McNally, 1997). As illustrated by the literature review and studies included in the current paper, objections to the concept of AS have been largely resolved (McNally, 1997).

Hypothesis: "High AS is strongly associated with fearfulness. People with high AS should hold fears of many different objects and situations, whereas people with low AS should relatively few fears."

Studies with healthy adults (Reiss et al., 1986), agoraphobics (McNally & Lorenz, 1987), and special populations (Haston & Stokes, 1987) have found high correlations between ASI scores and total scores on fear survey schedules. Since Reiss' (1991) review, several studies have provided further support for the hypothesis that AS is correlated with or predictive of common fears (Baranyai, 1991; Silverman, Fleisig, Rabian, & Peterson (1991); Taylor, 1993; Taylor & Rachman, 1992). Taylor (1996) provides further support. Using three separate samples (144 community volunteers, 135 patients with panic disorder, and 83 people with recurrent, unexpected panic attacks), Taylor found significant correlations between ASI scores and several common fears, including blood/injury fears, social fears, agoraphobia, and fear of animals.

Hypothesis: "AS is not found exclusively in agoraphobia"

It is clear now from numerous studies using nonclinical and community populations (i.e., Schmidt, Lerew, & Jackson, 1997) that AS is a normally distributed trait across populations. While high levels of AS are associated with anxiety pathology, high AS is not exclusively associated with disorder. Conversely, low levels of AS are not exclusive to nonclinical populations.

Hypothesis: "Some fears are motivated partially or wholly by expectations and sensitivities to anxiety"

Research analyzing the reasons underlying phobias and common fears has provided evidence supporting the hypothesis that fears can be motivated, at least in part,

by expectations of anxiety as opposed to only the expectation of external events (Reiss, 1991). Individuals with severe animal phobias have been found to fear panic attacks, insanity, embarrassment, and heart attacks upon encountering their phobic object (McNally & Steketee, 1985), suggesting that the object itself (i.e., the "external event") plays only a partial role in fear responses. Using factor analysis, Gursky and Reiss (1987) found that of 53 items on a scale measuring danger versus anxiety expectancies, 51 had factor loadings consistent with the distinction between danger and anxiety expectancies. There is little doubt about the existence of support for this hypothesis in general, but it seems that two areas have been neglected. First, while sensitivity has been the focus of research, the role of expectancy has received comparatively less attention. Second, research thus far has focused almost entirely on AS and largely neglected the other two sensitivities--illness/injury sensitivity and fear of negative evaluation.

The Role of Expectancies

Reiss' (1991) expectancy model of fear, anxiety, and panic holds that fear is a function of both expectations and sensitivities. Expectation refers to what the person thinks will happen, whereas sensitivity refers to the reasons a person holds for fearing and anticipated event. The role of expectations may be particulary relevant in the assessment and treatment of anxiety disorders, which are often (especially in the case of panic disorder and simple phobias) characterized by severe avoidance. There is speculation that expectations may be more important than sensitivities in determining avoidance.

Expectancy theory implies that the avoidance of a situation is determined by the interaction of danger expectancies and sensitivites. Therefore, since sensitivities (e.g.,

AS) are relatively stable, avoidance of situations within a given individual varies with that individual's expectancies. Data collected thus far (e.g., Craske, Sanderson, & Barlow, 1987) suggest that the expectation of panic among patients with panic disorder is strongly related to patterns of avoidance. In other words, in situations rated equally in terms of feared consequences where one is rated with higher expectation of panic occurrence, the situation rated for high expectation of panic is most likely to be avoided. Thus, although individuals with and without severe agoraphobia may be similar in terms of anxiety about the consequences of panic, those with severe agoraphobia may simply be more expectant of panic than others. In terms of treatment, this suggests that some patients may gain considerable benefit from a focus on expectancy modification rather that sensitivity modification per se.

While the concept of sensitivities is relatively uncontroversial, the theoretical role of expectancies in anxiety and panic is debateable. Psychological approaches to panic involve two inconsistent formulations of the role of expectancies (McNally, 1990). One view holds that Pavlovian conditioning applies to the learning and unlearning of expectancies in panic disorder. Learning is said to occur when one event provides new information about another event. Consistent with this view, increased anxiety expectancy has been shown to be associated with a decreased likelihood of panic. For example, patients informed about the effects of a carbon dioxide inhalation report less anxiety during inhalation than do uninformed patients (Rapee, Mattick, & Murell, 1986). In other words, patients who expect the bodily sensations associated with CO_2 inhalation exhibit less fear than do those who are surprised by such effects.

A contradictory view of expectancies however, holds that expectancies function as self-fulfilling prophecies, where increased expectation of panic actually increases the likelihood of panic (McNally, 1990). Evidence in line with this second formulation has also been found in challenge studies. Van den Hout and Griez (1982) manipulated subjects' expectancies by telling nonclinical subjects that an inhalation of CO_2 would produce either relaxation or tension. They found that subjects given relaxation expectations reported increased relaxation, while subjects given tension expectations reported increased tension.

Clearly, the role of expectancies in panic disorder is paradoxical. An important distinction between panic related events and those described by Rescorla and Wagner's (1972) theory may be partly responsible. In their original formulation, Rescorla and Wagner's relevant stimuli (e.g., tone and shock) lie outside the subject's control. In contrast, events relevant to panic (e.g., rapid heartbeat and full panic) are internally occurring events and therefore may be influenced by the subject's expectation (McNally, 1991). In other words, with panickers, expectations of panic may actually increase the probability of panic (i.e., self-fulfilling prophecy). Further investigation and clarification of the role of expectancies in panic is certainly warranted in order to resolve this discrepancy.

Other Sensitivities

Since the formulation of expectancy theory, AS has received a great amount of research attention and has been established as a meaningful personality trait with immediate implications for psychological health and possibly more distal implications for physical health. In light of the "success" of AS, illness/injury sensitivity and fear of

negative evaluation remain relatively unexplored. Earlier studies have however, established their validity. Similar to previously mentioned studies of anxiety sensitivity, studies using measures of injury sensitivity and the fear of negative evaluation have found correlations of approximately $\underline{r} = .5$ between these sensitivity scores and scores on fear survey schedules (Reiss, Peterson, & Gursky, 1988; Reiss, Altman, Belzer, Fetzer, Graves, & Kavesh, 1989). Even before Reiss' (1991) expectancy theory was completed, Watson and Friend (1969) found that the fear of negative evaluation accounted for 10-26% in variance of social fears, such as eating in public.

Taylor (1993) confirmed that the three fundamental fears (AS, injury/illness sensitivity, and fear of negative evaluation) are factorially distinct. These three fundamental fears accounted for 22-41 % of variance in common fears. Taylor suggests that the unexplained variance may be due to other fundamental fears that have not yet been identified or, more likely, due to idiosyncratic factors in fear acquisition (i.e., learning history, traumatic experiences). Apart from the main effects of fundamental fears and learning history, Taylor (1993) suggests that the intensity of common fears may be strongly influenced by the interactions among fundamental fears and the environment. In other words, fundamental fears would be ". . . most likely to exert their effects in the context of a history of aversive life experiences" (p. 297). For example, a person with intense illness/injury sensitivity may not fear dogs in the absence of an aversive experience with dogs. However, a phobia of dogs would be highly likely to develop should the person be bitten. Interestingly, of the three fundamental fears, Taylor (1993) suggests that fear of negative evaluation is the most important because it predicts general fearfullness and trait anxiety. Research has yet to shed light on this prospect.

Future studies should further the research that was begun on these other fundamental fears. Aside from finding more about the importance of AS, we may discover even more interesting and meaningful findings regarding the other fears alone or in conjunction with one another. The diathesis-stress model of anxiety pathology suggests that an individual with high levels of all three of these fears (i.e., a greater diathesis) would be at considerable risk for the development of psychological disorder, especially in the context of adverse life events. In addition, future research should consider the possibility of a hierarchical structure of fears and whether or not Reiss' (1991) three fundamental fears stem from higher order fears. Further elucidation of a comprehensive model of fears and fear acquisition is an exciting and likely valuable prospect in the arena of psychopathology research in addition to psychological treatment.

Are the Effects of AS Specific to Anxiety Pathology?

Anxiety and depressive disorders share many common etiological factors and the strength of these common factors may be more important than the strength of the etiological factors specific to each disorder (Andrews, 1996).

Recent evidence describes an association between anxiety sensitivity and depression (Otto, Pollack, Fava, Uccello, & Rosenbaum, 1995; Taylor et al., 1996) but this association has not been adequately evaluated. The present report (i.e., Schmidt, Lerew, & Joiner, 1998) suggests that anxiety sensitivity possesses symptom specificity with respect to anxiety pathology when a covariance strategy is utilized (i.e., examining changes in anxiety when controlling for changes in depression and vice versa). In general, these findings indicate that much of the association noted between anxiety sensitivity and depression is likely to be due to covariation among symptoms of anxiety

and depression. One question that may be raised is whether the symptom specificity criterion is too stringent because of the well-established overlap between anxiety and depression. Zero-order correlations among anxiety and depression measures attest to the substantial level of overlap in our sample (r range: .33 - .61). However, most views of anxiety and depression suggest that these are unique and discriminable phenomena. Prominent current views of psychopathology, such as the tripartite model (Clark & Watson, 1991) indicate that anxiety and depression have both overlapping and unique features including a factor specific to anxiety (i.e., physiological hyperarousal) as well as a unique depression factor (i.e., anhedonia). In this respect, the covariance strategy provides a means for partialling out the common features of anxiety and depression (i.e., negative affect) to better assess their unique features. In other words, anxiety sensitivity does not appear to act as a risk factor for the unique aspects of depression (e.g., anhedonia) but does appear to act as a risk factor for the unique aspects of anxiety (e.g., hyperarousal).

These findings are in keeping with anxiety sensitivity conceptualizations which predict that the key risk outcomes (i.e., panic attacks and panic disorder) for individuals with high anxiety sensitivity are perhaps quintessential representations of hyperarousal. This does not mean, however, that anxiety sensitivity does not amplify some of the associated symptoms of depression in the context of anxiety. Again, in terms of the tripartite model, anxiety sensitivity may amplify non-specific levels of negative affect but probably does not directly influence anhedonia. Further evaluations of anxiety sensitivity in the context of the tripartite model of anxiety and depression may elucidate some of these distinctions (cf. Joiner et al., 1998).

Andrews (1996) asserts that future studies of the etiology of individual anxiety and depressive disorders will need to be much more sophisticated than in the past. Specifically, he outlines three criteria that should be met in order to claim that a certain factor is specific to a particular disorder. First, the study should demonstrate that the factor is not typical of a normal nonclilnical population. Second, it should be shown that the proposed vulnerability factor is not present in a control group that is high in trait anxiety in order to demonstrate that the factor is not merely a "proxy measure" for factors such as trait anxiety. Finally, it should be shown that the factor does not occur in patients with other anxiety or depressive disorder, and therefore may be specific to the disorder being studied. More studies of specificity using several criteria will be necessary to confirm initial findings regarding AS.

The Anxiety Sensitivity versus Trait Anxiety Debate

One of the most important claims of the anxiety sensitivity hypothesis is that AS is conceptually and empirically distinct from trait anxiety (Lilienfeld, 1996). According to proponents of AS, trait anxiety is not sufficient to explain panic. Researchers continue to debate the distinctiveness of anxiety sensitivity and trait anxiety (Lilienfeld, 1996; McNally, 1996). According to proponents of AS, trait anxiety is not sufficient to explain panic. People vary in their proneness to anxiety, where some experience symptoms with little provocation and others only become anxious under the most anxiety provoking circumstances. Trait anxiety certainly measures the degree to which individuals differ in this anxiety proneness. Expectancy theory however, distinguishes between this vulnerability to anxiety symptoms and the propensity to fear these symptoms (i.e., anxiety sensitivity) (McNally, 1996).

The correlations of AS with trait anxiety indices typically range from $\underline{r}$ = .3 to .5. Lilienfeld (1998) argues that because AS is moderately correlated with trait anxiety, some of the findings attributed to AS might be attributable not to AS per se, but to the variance shared by AS with trait anxiety. For example, Lilienfeld et al (1989) suggest that findings demonstrating that agoraphobics obtain higher ASI scores than normals (Reiss et al., 1986; Taylor Koch, & McNally, 1992), that AS scores of agoraphobics return to normal limits following therapy (McNally & Lorenz, 1987) and that that AS moderates the association between mitral valve prolapse and panic disorder (Lyons, Talano, Titter, Martin, & Singer, 1986) are just as consistent with a trait anxiety explanation as they are with an AS explanation. Lilienfeld asserts that "although the ASI contains variance that is not shared with trait anxiety measures, it must be shown that this unique variance relates to the phenomena of interest" (e.g., panic attacks; p. 235).

Indeed, the ASI has been found to contribute information not provided by trait anxiety measures (Lilienfeld et al., 1993), and several longitudinal (e.g., Holloway & McNally, 1987; Maller & Reiss, 1992) and experimental (e.g, Rapee & Medoro, 1994) studies have demonstrated that the ASI posseses incremental validity above and beyond trait anxiety measures in the prediction of self-reported fears (Lilienfeld, 1998). Maller and Reiss (1992) evaluated whether state or trait anxiety was associated with panic attacks during the year preceding the follow-up evaluation (state and trait anxiety were assessed at follow-up). Both measures of state and trait anxiety were not found to be associated with the development of panic attacks. Moreover, the association between anxiety sensitivity and panic remained after statistically controlling for both state and trait anxiety.

People with high trait anxiety generally interpret a wide range of stimuli as threatening (Eysenck, MacLeod, & Mathews, 1987; Eysenck & Mathews, 1987; Phares, 1961). Thus, Lilienfeld (1996) suggests that trait anxiety alone may be sufficient for some people to respond fearfully to bodily sensations that are not inherently harmful. However, several findings by Rapee and Medoro (1994) suggest that this is not the case. In one study, Rapee and Medoro (1994) found that although two groups different in AS levels subjected to a hyperventilation challenge scored similarly on trait anxiety, the high AS group was more responsive than was the low AS group. If trait anxiety were the most important predictor of response, no differences should have been evident between groups (McNally, 1996). In two other experiments, Rapee and Medoro (1994) confirmed that ASI scores predicted variance in response to hyperventilation challenge above and beyond that predicted by trait anxiety. As pointed out by McNally (1996), this series of studies shows that unless also high in AS, individuals high in trait anxiety do not necessarily respond fearfully to bodily sensations.

Taylor, Koch, & McNally (1992) studied the relation between trait anxiety and anxiety sensitivity across the spectrum of anxiety disorders. They found that while mean trait anxiety scores did not differ among patients with social phobia, obsessive-compulsive disorder, posttraumatic stress disorder, and panic disorder, patients with panic dosrder had significantly higher ASI scores than patients with other anxiety disorders. Also, although panic and generalized anxiety disorder patients scored equally high in trait anxiety, patients with panic disorder scored significantly higher in terms of AS.

Findings reported in the current work (Schmidt, Lerew, & Jackson, 1997) indicate that the ASI accounted for a significant proportion of the variance in the occurrence of anxiety pathology after controlling for the effects of trait anxiety. Trait anxiety did not predict panic. Interestingly, entry of trait anxiety into the regression equation decreased the strength of association between the ASI and panic. This suppression effect (Cohen & Cohen, 1983, p. 95) suggests that controlling for trait anxiety removes criterion-irrelevant variance from anxiety sensitivity, i.e., it appears that the "part" of anxiety sensitivity that is not associated with trait anxiety is especially relevant to the criterion (panic). We also evaluated Lilienfeld et al.'s interpretation of expectancy theory which suggests that the development of panic may be potentiated when high trait anxious individuals also possess high anxiety sensitivity. The failure to find significant anxiety sensitivity by trait anxiety interactions suggests anxiety sensitivity and trait anxiety do not act synergistically to predict the development of panic. In sum, these analyses offer an important demonstration that anxiety sensitivity possesses exhibits incremental validity above and beyond triat anxiety. Accordingly, the ASI possesses reliable variance that is not shared with trait anxiety measures (Lilienfeld et al., 1993; McNally, 1996).

The argument then, appears to have evolved from debate over whether or not AS is trait anxiety to debate as to how AS and trait anxiety are interrelated. It is possible that the fear of anxiety symptoms (i.e., AS) stems from a higer order proneness to experience anxiety (i.e., high trait anxiety) (Schmidt, 1998). Lilienfeld et al. (1993) argue that the key to resolving the AS vs. trait anxiety argument lies in distinguishing among different levels of trait specificity and generality. Specifically, Lilienfeld, Turner, and Jacob (1993) propose that AS may be a lower-order dimension of trait anxiety. According to this

hierarchical model, trait anxiety may be considered as the tendency to react anxiously to anxiety producing stimuli in general, whereas anxiety sensitivity is a more specific proneness to react anxiously to one's own anxiety related symptoms (Lilienfeld, 1996). So AS would share variance with trait anxiety but would also possess unique variance unrelated to trait anxiety. The other sensitivities of expectancy theory (i.e., injury sensitivity and the fear of negative evaluation) may also exist as distinct factors (on the same order) within the model. Therefore, according to such a model, AS provides information distinct from trait anxiety, but is still conceptually and empirically related to trait anxiety. Lilienfeld (1996) posits that this hierarchical model of anxiety helps to explain four consistent findings: A) the positive correlation between the ASI and trait anxiety measures, B) findings of incremental validity of the ASI relative to trait anxiety measures, C) the finding that some people exhibit high AS and low trait anxiety (and vice versa), and D) the finding that panic disorder patients exhibit levels of trait anxiety similar to patients with generalized anxiety disorder but have higher levels of AS (McNally et al., 1991).

As this hierarchical model becomes more widely accepted, it will surely bring with it implications for future research and our understanding of the nature of anxiety. One crucial research implication of hierarchical models is that higher- and lower-order dimensions can provide competing explanations for certain hypotheses (Watson & Clark, 1992). For example, if a hypothesis is proposed concerning the relation of a lower-order dimension to external criteria, but a measure of the higher-order dimension on which this lower-order dimension loads is not included, it may be incorrectly concluded that the hypothesis has been confirmed. In other words, the observed relation may in fact be

attributable to the influence of the unmeasured higher-order dimension. It is important then, that future AS research also examine the extent to which AS relates to trait anxiety (Lilienfeld, 1998). In addition, an emphasis to date has been on determining how AS differs from trait anxiety, and future studies would do well to investigate the similarities beween AS and trait anxiety. A knowledge of both questions will almost certainly provide important information regarding not only the etiology of anxiety disorders, but of AS itself (Lilienfeld, 1998).

Anxiety Sensitivity Subdomains

AS was originally proposed as a unitary construct (Reiss & McNally, 1985). Empirical evaluation of this proposition using the ASI has yielded somewhat inconsistent findings. Several early factor analytic studies provided support for the unitary nature of AS (Reiss, Peterson, Gursky, & McNally, 1986; Taylor, Koch, & Crockett, 1991). Others found that the ASI is multifactorial (Telch, Shermis, & Lucas, 1989; Wardle, Ahmad, & Hayward, 1990). There appears to be a growing consensus, however, which indicates that the ASI is hierarchical (i.e., best regarded as unifactorial at a higher level but multifactorial at a lower level) and composed of three first-order factors measuring fears of adverse physical outcomes (Physical Concerns), fear of cognitive incapacitation (Mental Concerns), and fear of publicly observable symptoms (Social Concerns) (Cox, Parker, & Swinson, 1996, Stewart, Taylor, & Baker, 1997; Zinbarg, Barlow, & Brown, 1997). First-order AS factors have been found to differentially predict fearful responding to a biological challenge involving inhalations of high concentrations of CO_2 (Schmidt, Lerew, & Jackson, in press) but these lower-order factors have not been evaluated as predictors in the context of a prospective study (Schmidt, 1998).

There is some evidence to suggest that AS subfactors may differentially predict anxiety. Further analysis of the USAFA data presented in Schmidt, Lerew, and Jackson (1997) indicates that only the Mental and Physical Concerns subscales of the ASI were important predictors of panic and anxiety symptoms during BCT. Although we had expected individuals scoring high on the Physical Concerns factor to be at greatest risk for panic due to the physical stressors inherent to basic training, the Mental Concerns factor proved to be the only subfactor that predicted panic after controlling for history of panic and trait anxiety. Upon reflection, it is clear that the mental stressors, compared to the physical stressors, associated with basic training are likely to be relatively more novel and challenging. Future studies may determine whether the Mental Concerns component of anxiety sensitivity is a generally important predictor of panic in other contexts, or whether the relationship found in this recent study is largely due to study-specific factors such as the novelty of the stress. The biological challenge paradigm offers an alternative methodology for examining the role of these subfactors in the genesis of panic. If the Mental Concerns subcomponent of anxiety sensitivity is a generally important predictor, it would be expected to consistently outperform the other subfactors and/or the overall ASI in predicting panic to various challenge agents (e.g., 35% carbon dioxide, cholecystokinin tetrapeptide, sodium lactate).

Conclusion

To date, few published reports exist that have prospectively examined the onset of anxiety and panic in nonclinical populations (Balon, Pohl, Yerigani, Rainey, & Berchou, 1988; Breslau & Davis, 1993; Eaton & Keyl, 1990). Even fewer exist that have prospectively examined the role of anxiety sensitivity in anxiety pathology (Ehlers, 1995;

Maller & Reiss, 1992; Schmidt, Lerew, & Jackson, 1997). These few studies however, have all provided firm evidence that anxiety sensitivity is a risk factor, for the development of anxiety pathology rather than just a side effect of elevated anxiety. Considering the difficulty that is often encountered in longitudinal studies (obtaining a large sample, retaining subjects), such studies may remain relatively rare. The base rate for panic attacks and anxiety disorders presents one major problem for longitudinal studies. The annual incidence of panic disorder has been found to be approximately two percent, and the incidence for panic attacks approximately ten percent (Keyl & Eaton, 1990). Because clinical anxiety and panic attacks occur so infrequently, longitudinal studies are at risk for lacking appropriate levels of power to detect meaningful data (Schmidt, 1998). However, prospective studies are important in that they provide important checks on ecological validity in regard to the development of anxiety and panic (and other psychopathology) outside the laboratory (Schmidt, 1998).

The observed level of variance in anxiety and panic accounted for by anxiety sensitivity strongly suggests that there are other important factors involved in the onset of anxiety. Factors generally outside our realm of control such as sex, age, and negative life events have been shown to increase the risk for the development of anxiety. These factors may independently contribute, or interact with personality variables such as anxiety sensitivity, to predict the development of psychopathology. From the epidemiological perspective, personality vulnerability is a necessary cause of anxiety symptoms in the general population (Andrews, 1996). In one population study, Duncan-Jones (1987) concluded that short term stressors were of relatively little importance in comparison to long term vulnerability factors such as neuroticism (Eysenck & Eysenck, 1975). In this

study, personality traits accounted for 70-76 % of the variance in symptoms. However, as illustrated in the USAFA data, the diathesis-stress model likely presents the best model for the pathogenecity of anxiety. Anxiety pathology often emerges during highly stressful times (Pollard, Pollard, & Corn, 1989). Faravelli and Pallanti (1989) reported that panic disorder patients, compared to nonclinical controls, experienced significantly more negative life events during the year preceding the development of panic, and that the majority of these negative stressors occurred in the month preceding panic (Schmidt, 1998).

Other possible vulnerability factors (e.g., body vigilance) have received only minimal attention and are just beginning to become a part of longitudinal anxiety research (Schmidt, Lerew, & Trakowski, 1997). Support for the pathogenicity of these variables, as presented in the current series of studies, also looks promising. Constructs similar to anxiety sensitivity such as body vigilance and discomfort intolerance are likely to be more closely scrutinized in the future as researchers attempt to finely dissect the structure of anxiety. Parameters such as predictability and control (Barlow, 1988) that have also been shown that affect fear and the development of panic also hold promise.

Preliminary research in our lab has indicated two important findings. First, it appears that anxiety sensitivity can indeed be reduced in a nonclinical population. In one sample of nonclinical individuals, a brief cognitive intervention (i.e., approximately twenty minutes of education about the nature, causes, and implications of anxiety sensitivity) appeared to be effective in reducing anxiety sensitivity. In light of the findings presented in the above studies and suggestions that primary prevention anxiety interventions may be possible, this finding is especially promising.

Interestingly, a second finding regarding AS reduction in nonclinical individuals has emerged. Similar reductions in AS have been observed in individuals receiving a brief *non-specific* cognitive intervention (i.e., approximately twenty minutes of education about *the nature, causes, and deleterious effects of stress in general*) that did not emphasize the benign nature of bodily sensations or even mention the construct of anxiety sensitivity. It appears that even simple, broad-based "stress management" interventions (as opposed to interventions specifically designed to address anxiety sensitivity) may be sufficient to achieve reductions in AS. Well-controlled empirical studies of the nature of cognitive treatment required are warranted to investigate this question. Should it be possible to reduce AS with a simple, brief, easily administered, and inexpensive stress education protocol, clinicians and even paraprofessionals may be in a position to prevent the onset of maladaptive anxiety in many at-risk population. Cadets at USAFA are just one example of individuals who may be easily "innoculated".

Findings suggesting that AS need not be specifically addressed in order to be reduced lead to a larger theoretical issue concerning the necessity of addressing underlying cognitive variables whatsoever in the treatment and prevention of anxiety and panic. Certainly, medication alone (e.g., Clonazepam, a high-potency benzodiazepine) has received the endorsement of many clinicians and experts, including those scientists who participated in the National Institutes of Health Consensus Conference on the Treatment of Panic Disorder (National Institutes of Health, 1991). Despite a general level of endorsement, clinicians vary in their emphasis on drug treatment (McNally, 1994). Some clinicians assert that medication is essential for blocking panic and that behavioral interventions are necessary only for agoraphobics who are unable to perform self-

exposure to feared situations. Some consider medication only after a failure in response to cognitive and behavioral treatments, and others ascribe to a general regimine of combination therapy (both medication and cognitive-behavioral therapy) (McNally, 1994). Further complicating the issue of AS reduction are observations of AS reductions following medication treatment of panic disorder (Otto & Reilly-Harrington, 1999). For example, Otto, Pollack, Sachs, and Rosenbaum (1991) found a significant reduction in ASI scores in patients who were treated with benzodiazepines alone. However, studies of the efficiency of pharmacotherapy for panic disorder have not typically included the ASI as a measure, "making it difficult to draw conclusions about the relative impact of psychosocial compared with pharmacological treatments on AS" (p. 332; Otto & Reilly-Harrington, 1999). Medication treatment alone can lead to significant reductions in AS, but ASI scores remain elevated in medication treated patients with residual symptoms (Bruce, Spiegel, Gregg, & Nuzzarello, 1995). In such cases, cognitive-behavioral therapy offers an effective strategy to further decrease AS and to aid medication discontinuation (Hegel, Ravaris, & Ahles, 1994). Given the complexity of some presentations of anxiety, combining treatment modalities is warranted in many cases (Telch, 1994).

If a vulnerability factor is a major cause of liability to anxiety, then treatment programs should attempt to reduce this risk factor in addition to reducing symptoms. In this way, a major advance may be taken in the prevention of relapse (Andrews, 1996). Treatment efficacy can be improved by identifying and measuring changes in risk factors such as anxiety sensitivity. As of now, a strong body of research has been amassed supporting the efficacy of cognitive behavioral therapy in reducing vulnerability factors. Future studies should provide tests not only of the effects of vulnerability factors on the

development of pathology, but also of methods to reduce these factors in both clinical and nonclinical populations.